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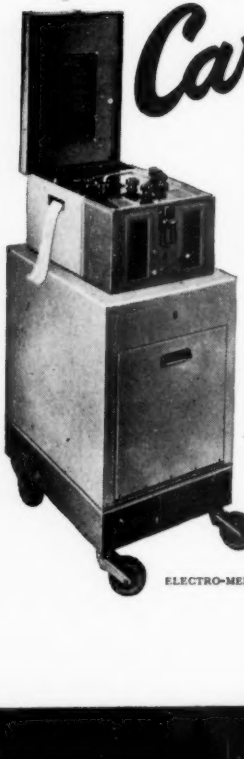

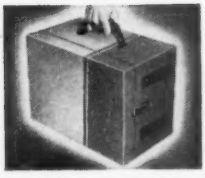

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Section of Ophthalmology

President—Professor W. J. B. RIDDELL, M.D., F.R.F.P.S., F.R.S.Ed.

[*March* 12, 1953]

The Ocular Troubles of Dr. Johnson and Mr. Pepys

By P. H. BEATTIE, M.D.
Norwich

I HAVE chosen Dr. Johnson as the subject of my Address because I find him a fascinating personality, and also because, as far as I know, no oculist has written about his eye troubles. Some distinguished general physicians and surgeons have considered his general health, but their remarks about his vision have sometimes been very wide of the mark.

Samuel Pepys:

I will first, however, briefly mention Samuel Pepys. His visual troubles have been discussed in detail by Sir D'Arcy Power, Mr. R. R. James, and others, but not in recent years. Samuel Pepys' Diary records only ten years of his life. He wrote his last words in this Diary when he was 36 years old—with great sorrow, for failing sight had obliged him to discontinue and he believed he was destined to become blind, like his great contemporary Milton. Early in the Diary, in 1660, Pepys refers to an injury to his right eye whilst firing a cannon in the Royal Salute to Charles II, but the injury does not appear to have been serious, and there is no further reference to it; two main symptoms recur: photophobia and inability to read for more than a few minutes. Candles at the theatre gave him "mighty pain". Pepys realized that alcoholic excesses aggravated the inflammation and photophobia. On one occasion he thought a change of brewer caused his discomfort. He tried green spectacles and oiled paper and had an eye shade made to protect his eyes. He used lotions which made his eyes "smart horribly" and, according to the fashion of the times, was relieved of 14 oz. of blood.

His inability to read and write formed his most serious difficulty. He dictated his letters for a time and got through his business in this way. His wife read to him in the evenings. He consulted Turlington, the great spectacle maker, who persuaded him not to use "old spectacles"—that is, convex lenses "magnifying much" such as would be used for presbyopia. Turlington evidently thought that a man of 34 should not be presbyopic and that convex lenses would be harmful. Pepys himself noted when he was 35 "I am come that I am not able to read out a small letter, and yet my sight good for the little while I can read, as it ever was, I think". In 1668, he records: "The month ends mighty sadly with me, my eyes being now past all use almost; and I am mighty hot upon trying the late printed experiment of paper tubes." These experiments were published under the title of "An easy help for decayed sight". The inventor of these tubes was a man of 60 and from the description of his symptoms I think he probably had nuclear cataracts. He found that by using tubes 3 in. or so long, with a small orifice at the lower end sufficient to admit the tip of the little finger, he could read satisfactorily. The tubes presumably reduced the light entering the eye, permitted dilatation of the pupil and enabled him to see round the central opacity. Pepys did try the tubes, with some success at first, but he became discouraged later and abandoned his Diary.

The question at issue is—what sort of refractive error caused these symptoms? Sir D'Arcy Power suggested that Pepys had a hypermetropic astigmatism, which could not be corrected by the optical knowledge of the time. But this would not explain how Pepys could see very well when he started to read, nor how a monocular tube improved his vision temporarily; nor, indeed, how he was able to see well enough to write his Tangier Journal fourteen years later and to spend a life in study and official duties. Mr. R. R. James has suggested that he had convergence deficiency and might have benefited from base-in prisms; Leslie Paton, I believe, thought he had hyperphoria. I would suggest that he had hypermetropia with esophoria. A monocular tube would have helped him temporarily, but he was unable to sustain the accommodative effort with one eye. Later, when, perhaps, Mr. Turlington considered he might safely wear a presbyopic correction, his esophoria was relieved by strong convex lenses and he was able to continue his duties and studies till he died at the age of 70.

Samuel Johnson:

On Monday, May 16, 1763, when Boswell first met Samuel Johnson, he wrote in his Diary: "Mr. Johnson is a man of most dreadful appearance. He is a very big man, is troubled with sore eyes, the palsy and the king's evil." Johnson, at this time, was 54 years old, but his eyesight had been a source of trouble from his earliest years. He tells us in his fragment of autobiography that he was taken home from his nurse "a poor diseased infant almost blind" and in his second year was taken to consult a Worcester oculist, Dr. Atwood, but we do not know what disease was diagnosed nor the treatment prescribed. Later, when he was 2½ years old, on the recommendation of Sir John Floyer, he was taken from Lichfield to London by stage coach to be "touched" by Queen Anne for his scrofulous infection. He was one of two hundred at this ceremony. As Macaulay says, "he was inspected by the court surgeon, prayed over by the court chaplains and stroked and presented with a piece of gold by Queen Anne". The piece of gold given to him may be seen in the British Museum to-day. (The speaker here exhibited a similar "touch piece" which Mr. Charles Noon, the senior surgeon at the Norfolk and Norwich Hospital had kindly lent him.) But the treatment was ineffective. He bore the scars of his tuberculous adenitis to his dying day. Witnesses differ on the disfigurement caused by this: Bishop Percy describes a simple scar under one of his jaws which "excites no disgust", but Mrs. Thrale declares that the affliction left "such marks as greatly disfigured a countenance naturally harsh and rugged". The scars were probably tolerably evident, otherwise Boswell would not have noted them on his first meeting at Mr. Davis's bookshop.

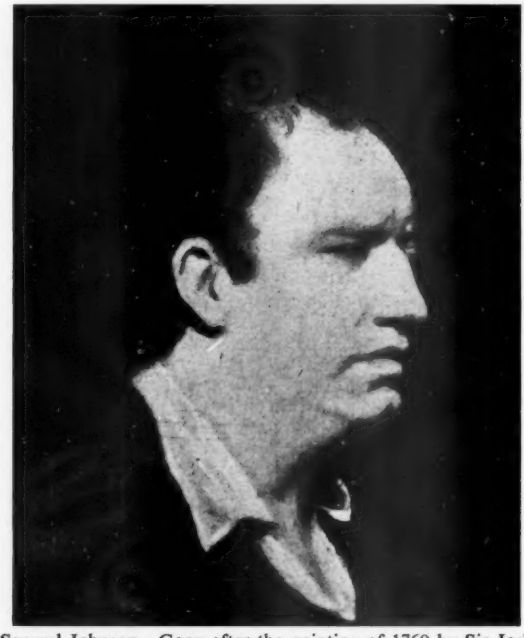

FIG. 1.—Dr. Samuel Johnson. Copy after the painting of 1769 by Sir Joshua Reynolds. *National Portrait Gallery copyright.* By kind permission.

The expression used by Boswell, "sore eyes", suggests, I think, a chronic blepharo-conjunctivitis. Probably this inflammation was present in infancy and childhood in a more acute phase and as it is generally agreed that he suffered from a genuine scrofulous infection, it is justifiable to suppose the condition was a phlyctenular conjunctivitis, possibly with a complicating keratitis as well. Such an early history, with an occasional secondary infection, would cause chronic redness of the lid margins and conjunctival injection and might well merit the description of "sore eyes". The appearance of the eyes in Reynolds' portrait in the National Gallery, painted in 1769, suggests a chronic blepharo-conjunctivitis (Fig. 1). However, we are told by Birkbeck Hill that this is probably an idealized picture: it represents Johnson in the prime of life, between 40 and 50, whereas it was painted when he was 60. Also, it must be admitted that other portraits of Johnson, even those painted by Reynolds, show eyes wide open and apparently normal.

If we grant that Johnson's eyes had some signs of chronic superficial inflammation in actual life, there was no noticeable difference between the two organs. But we know that one eye had very poor vision indeed. Johnson himself, speaking to Dr. Burney, said characteristically "the dog was never good for much". Boswell tells us that "he did not see with one of his eyes, though its appearance was little different from that of the other". Mrs. Thrale says "one eye was perfectly useless to him; that defect, however, was not observable, the eyes looked both alike". Hawkins and Arthur Murphy tell us it was his left eye. We have no satisfactory or conclusive evidence concerning the cause of this poor vision. It may have been a highly myopic eye or a markedly astigmatic one which, as a result, became severely amblyopic early in life. Possibly the scarring of phlyctenular keratitis destroyed useful central vision. However, enough vision seems to have been retained to keep the eye in good alignment, as none of his portraits show any tendency to a divergent strabismus. Even the deeply impressive Rembrandt-like picture by Reynolds of Johnson in old age portrays a pair of wistful but very straight eyes (Fig. 2).

FIG. 2.—Samuel Johnson in old age. From the painting by Sir Joshua Reynolds, *c.* 1782–4, *in the possession of Miss Caroline Newton.* By kind permission.

However, the good eye was not a wholly satisfactory servant; it seems to have been short-sighted throughout his life. Johnson himself acknowledged this on numerous occasions, but when Dr. Percy said to him "but, my good friend, you are so short-sighted and do not see as well as I do" Johnson was furious. "Hold, Sir", he said, "don't talk of rudeness. Remember, Sir, you told me I was short-sighted. We have done with all civility. We are to be as rude as we please." But Johnson protested too much. Fanny Burney, who loved Johnson exceedingly, says "Notwithstanding he is so absent, and always so near-sighted, he scrutinizes into every part of almost everybody's appearance", but she added that he could see wonderfully at times. Murphy, an old friend of Johnson, tells us that Garrick was "hurt that his Lichfield friend did not think so highly of his dramatic art as the rest of the world. The fact was Johnson could not see the passions as they rose and chased one another in the varied features of that expressive face". Murphy also records that one day whilst sitting at table with the celebrated Mrs. Cholmondeley "Johnson took hold of her hand in the middle of dinner and held it close to his eye, wondering at the delicacy and whiteness, till with a smile she asked: 'will he give it to me again when he has done with it?' " Reynold's portrait of 1775 shows Johnson reading with the bent-up book close to his face (Fig. 3). This is probably the picture which aroused Johnson's displeasure, provoking him to protest to Mrs. Thrale that "he would not be known by posterity for his defects only, let Sir Joshua do his worst". He added that Reynolds "could paint himself as deaf if he chooses, but I will not be blinking Sam". The word "blink" here as defined by Johnson in his

dictionary, means "to see obscurely". (A "blinkard" was one who had bad eyes.) This picture was painted when Johnson was 66 and if we assume that his lifelong defect was uncomplicated myopia and allow for the normal presbyopic changes, it suggests that he probably had somewhere between four and five dioptres of myopia. However, there are certain objections to this assumption. With this refractive error his distance vision would have been very poor indeed and he could not have corrected Boswell about the precise shape of a distant hill, as he did on his Hebridean tour; also, at Versailles, when he was 66, his observations were remarkably precise. For example, he records in his journal that "Amongst the birds was a pelican, who, being let out, went to a fountain, and swam about to catch fish. His feet well webbed: he dipped his head and turned his long bill sidewise. He caught two or three fish but did not eat them". It is doubtful if he could have made such an observation with a myopic error of four or five dioptres, even by half closing his eyes, also he would not have felt obliged to tell Garrick "I'll come no more behind your scenes, David, for the silk stockings and white bosoms of your actresses excite my amorous propensities".

FIG. 3.—Samuel Johnson. From the painting by Sir Joshua Reynolds, *c.* 1775, *in the possession of the Hon. Mrs. Arthur Guinness.* With acknowledgments.

An uncomplicated myopia could have been corrected by spectacles, but there is no picture of Johnson in glasses and no references to their use by Johnson in Boswell or other memoirs.[1] He himself had no prejudice against their use, but he realized their limitations. He quotes in his dictionary "It is no fault in the spectacles that the blind man sees not", and a short verse by Swift:

> "This day then let us not be told
> That you are sick and I grown old
> Nor think on our approaching ills
> And talk of spectacles and pills."

In a characteristic piece of prose in the "Lives of the Poets" he condemns Swift for not wearing spectacles:

> "Having thus excluded conversation, and desisted from study, he had neither business nor amusement; for having by some ridiculous resolution or mad vow, determined never to wear spectacles, he could make little use of books in his later years: his ideas, therefore, being neither renovated by discourse, nor increased by reading, wore gradually away, and left his mind vacant to the vexations of the hour, till at last his anger was heightened into madness."

His friend Reynolds wore glasses frequently. Simple concave lenses were in common use at any rate on the Continent. As early as 1519 a picture by Raphael shows Pope Leo X (Giovanni de Medici) holding a quizzing glass and the reflections suggest the lens was concave. This Pope was known to be short-sighted and is said to have worn a monocle whilst hunting. Herr Ahlström, of Stockholm, recently published a paper[1] describing a pair of spectacles fitted with lenses of power −6·50 spheres on each side which were presented by the city of Augsberg to King Gustavus Adolphus in 1632. Herr Ahlström tells me he possesses a printed notice issued by the opticians P. & J. Dollond in St. Paul's Church-Yard dated 1766 advertising "concave glasses for short-sighted persons". It seems inconceivable that Johnson, who was keenly interested in science and medicine and could discuss the action of lenses with King George III, did not know about the use of glasses for myopia. We can only presume that he tried them and found them of no advantage. The reason for this may be that his short sight may not have been ordinary axial myopia. He may have had a diffuse, shallow and invisible scarring of his cornea, a legacy of his phlyctenular kerato-conjunctivitis, possibly with very small clear areas which minimized the effects of presbyopic changes. It is possible also that he had a high degree of myopic astigmatism and this could not be corrected by the optical knowledge of the time.

His relatively good eye was subject to attacks of inflammation. In 1756, when he was 47, he wrote a prayer on February 15: "Almighty God Who has restored light to my eye, and enabled me to pursue again the studies which Thou hast set before me . . ." But the trouble recurred and four days later he wrote: "The inflammation is come again into my eye, so that I can write very little." Seventeen years later, at the age of 64, whilst learning Low Dutch, he developed a fever and this left him, as he wrote to Mrs. Thrale, with a very severe inflammation in the seeing eye. His vision was so darkened that for a time he could not read by it. His friend Barretti evidently discussed this attack with Dr. John Mudge, a physician of their acquaintance: he did not suspect glaucoma but he considered the inflammation very severe and threatening the vision. Johnson tells us the inflammation was successfully treated by two copious bleedings and the prolonged use of cathartics. He adds, however, that two months after the attack "the effect yet remains". Presumably the recovery was not absolutely complete, but it was sufficient to allow him to embark on the three months' Tour of the Hebrides a few weeks later in August 1773. He talked a great deal on this tour and made many a pungent comment, but we hear no complaint about his eye or vision. Indeed, we do not hear again during his lifetime of any eye trouble—i.e. during the last eleven years of his life. It is interesting to speculate about his two severe attacks of inflammation. He may have had a corneal ulcer at the site of his phlyctenular scarring, but unless this were central (which is unlikely) his vision would not have been darkened. The onset of the inflamed eye after a fever suggests possibly a dendritic ulcer which lingered for a month or two and left a slight residual opacity. He may have had an iritis, as Sir Russell Brain suggests. Johnson suffered from attacks of rheumatism and gout and might therefore seem predisposed to uveal inflammation. He describes his inflammation as severe, but we do not hear complaints of pain either in the eye itself or in the frontal or temporal region. Johnson was the victim of a severe neurosis, but his attitude to pain was stoical. When Dr. William Heberden and Dr. Mudge advised excision of his sarcocele—Johnson was then an old man of 74—his attitude was one of the most commendable fortitude. He said he would endure pain with decency if there was a danger of gangrene and added to Mrs. Thrale: "You would not have me for fear of pain perish in putrescence." His lack of complaints, therefore, does not necessarily mean no pain was experienced. The true nature of this inflammation remains obscure.

Although Johnson numbered many distinguished doctors as friends and was himself very interested in medical theory and practice, he did not apparently consult his contemporary, the renowned Chevalier Taylor. He had met this prince of medical charlatans, this "ophthalmiator Pontifical, Imperial and Royal" as he called himself, but Johnson considered him the most ignorant man he had ever known, probably because he could not converse in Latin. He quoted him as an instance of how far impudence could carry ignorance. Johnson was quite prepared to discuss ocular troubles and evidently took an interest in the subject of cataract, probably in part because Anne Williams, who occupied part of his house in Gough Square and Bolt Court, was blind from this disease. In 1760, when he was 51, we find him writing to his friend Bennett Langton in Lincolnshire about cataract in words which are strangely appropriate to-day, although they refer to the operation of couching practised at that time:

"I am very sincerely solicitous", he says, "for the preservation or recovery of Mr. Langton's sight, and am glad that the chirurgeon at Coventry gives him so much hope. Mr. Sharpe is of opinion that the tedious maturation of the cataract is a vulgar error and that it may be removed as soon as it is formed. The notion deserves to be considered. I doubt whether it be universally true, but if it be true in some cases, and those cases can be distinguished, it may save a long and uncomfortable delay."

Johnson's medical history and the post-mortem findings suggest that he suffered from hypertension, renal disease and cardiac failure, but he evidently had no retinal complications affecting the macular area. No noticeable deterioration of vision occurred, even in his later years. Reading and writing

[1] AHLSTRÖM, O. (1951) Two pairs of spectacles from the Art Cabinet of Gustavus Adolphus, *Opt. sci. Instr. Mkr.*, **122**, 595.

letters were the consolations of old age when loneliness and suffering oppressed him. The words of his letters a week or so before his death are well formed and the layout is obviously the work of a man whose eyesight is satisfactory. It would seem that his right eye, though poor, was adequate and enabled him to sustain the prodigious labours of his dictionary and a wide and miscellaneous variety of reading. His defective vision may have been responsible for his ability to seize the substance of a book and judge its quality without laboriously reading it through. Also, it may have restricted his interest in the visual arts of painting and architecture, as deafness made him unresponsive to music. But these restrictions canalized his powerful intellect into the study of men and morals and the main vehicle of his later years was his talk. He gave much sensible and pointed advice: he tells us in Rambler No. 19 that the profession of physic demands "melancholy attendance upon misery, mean submission to peevishness, and continual interruption of rest and pleasure". But he reminds us elsewhere that a man is seldom so innocently employed as when earning his own living. We can take some consolation from this.

The Later Results of Perforating Eye Injuries

By Mary Savory, F.R.C.S.

This paper is based on a study of 144 consecutive cases of perforating eye injury treated at one branch of the Royal Eye Hospital, London, between August 1945 and June 1949. Results are only classified as known if the case has been followed for over three and a half years. The cases were under the care of all the surgeons of the hospital, and received treatment on the generally accepted lines.

The method of follow-up was, firstly, to analyse the in-patient and out-patient notes. Letters were then sent to all patients who had ceased attending. Patients who had retained the injured eye were asked to attend hospital by special appointment, but if this was impossible, to answer a few simple questions about the injured eye. Only 5 cases used the questionnaire, and in 2 of these it was considered that the visual results could not be accurately assessed and they are classified as unknown. I have examined those patients who came to hospital specially, and have used the out-patient notes of those who were still attending. Patients who lost the injured eye were sent a short questionnaire asking about the health of its fellow. Of the 105 retained eyes, 79 were followed for more than three and a half years, some for as long as seven and a half years. Of the 39 patients who lost an eye, 29 have replied or recently attended hospital.

Overall results (Table I).—In these tables anterior segment injury implies injury to the anterior chamber with or without iris damage. Posterior segment injury includes wounds of the ciliary region. It is part of the recognized teaching that injury to the lens much increases the severity of a perforating wound and, as this has been fully borne out in this series, the results have been tabulated according

Table I.—Overall Results : 144 Cases

Injury	No. of cases	Result known	Excised	6/9 or better	6/12 to 6/24	6/36 to 4/60	<4/60	Result unknown
Lens Undamaged: 78 Cases								
Anterior segment	39	29	4	18	5	—	2	10
Posterior segment	24	20	7	9	1	1	2	4
Anterior and posterior segments	15	10	2	4	1	—	3	5
Localized Lens Opacity: 17 Cases								
Anterior segment	12	11	—	7	3	1	—	1
Anterior and posterior segments	5	4	—	2	2	—	—	1
Traumatic Cataract: 49 Cases								
Anterior segment with or without posterior segment	49	44	26	6	—	2	10	5
Totals	144	118	39	46	12	4	17	26

to whether the lens was damaged or not: of the 79 eyes retained and followed for over three and a half years, 46 (58%) had vision of 6/9 or better, and in cases without traumatic cataract the good vision rate rises to 65%. On the other hand 39 eyes came to excision, a rate over the whole series of 27%. No case of sympathetic ophthalmia was found.

Reasons for excision of the injured eye (39 *cases*).—3 were removed immediately because the wound had disorganized the globe beyond hope of repair. In 6 a retained foreign body set up inflammation leading to enucleation. Infection necessitated the early removal of 6 eyes, and 1 was excised for rupture of the wound with secondary hæmorrhage and ciliary prolapse. 4 were enucleated for secondary glaucoma. The 19 remaining eyes were removed because of inflammatory changes which I have classified as follows: endophthalmitis 7, uveitis 6, iridocyclitis with risk of sympathetic ophthalmia 6.

The endophthalmitis group may contain cases where there was a mild or partly controlled infection. It was only during the last two years of this period that big doses of penicillin were being given by subconjunctival injection, and streptomycin was not used. There was a slightly lower excision rate for the last two years, but it is not statistically significant. There is indeed little hope that increased use of the antibiotics can make a major difference to the excision rate as only relatively few eyes are lost from sepsis.

Time of excision of injured eye.—Table II shows that more than three-quarters were removed during the first six weeks.

TABLE II.—TIME AT WHICH EYES WERE EXCISED

Immediate	3	= 31 eyes	During 3rd month	3	= 8 eyes
During 1st week	6		During 4th month	2	
During 2nd week	7		During 5th month	1	
During 3rd week	5		During 6th month	1	
During 4th week	2		At 9 months	1	
During 5th week	4				
During 6th week	4				

Total 39 eyes

Intra-ocular foreign bodies.—This series contained 66 cases of intra-ocular foreign body, all of which were due to ordinary civilian accidents.

(*a*) *Foreign body removed* (Table III*a*).—A magnetic foreign body was removed in 56 cases. The anterior route was used for foreign bodies in the anterior chamber, and for those in the posterior segment if traumatic cataract was present. With 2 exceptions, the posterior route was used for those behind the ciliary body if the lens was clear. In 4 cases the foreign body was removed through a limbal wound of entry.

As would be expected, the anterior route, where the lens was clear, has given good results in the cases traced. The one poor result had siderosis before operation. Unfortunately several cases could not be followed up, but the known results do not differ materially from those of similar wounds without an intra-ocular foreign body. The posterior route has also given good results. So-called retinitis proliferans has only occurred in 3 cases, and in these the foreign body was situated far back, and the incision for removal was made at or behind the equator. The results when traumatic cataract was present were not good, but it will be shown that the traumatic cataract, not the foreign body, is responsible for this.

4 cases in which siderosis was present before removal of the foreign body have not done well: 2 had bad results at two and a half years and have ceased attending; the vision in the remaining 2 eyes is 6/24 and counting fingers.

TABLE III.—RESULTS OF INTRA-OCULAR FOREIGN BODIES

(*a*) FOREIGN BODY REMOVED: 56 CASES

Route of removal	No. of cases	Result known	Excised	6/9 or better	6/12 to 6/24	6/36 to 4/60	<4/60	Result unknown
WITHOUT TRAUMATIC CATARACT								
Anterior	18	9	—	6	2	—	1	9
Posterior	16	13	2	7	2	1	1	3
Through limbal entry wound	2	2	2	—	—	—	—	—
WITH TRAUMATIC CATARACT								
Anterior	18	15	10	3	—	—	2	3
Through limbal entry wound	2	1	1	—	—	—	—	1
Totals	56	40	15	16	4	1	4	16

(*b*) FOREIGN BODY NOT REMOVED: 10 CASES

	Excised	6/9 or better	6/12 to 6/24	6/36 to 4/60	<4/60
Non-magnetic	4	—	—	—	—
Magnetic or probably magnetic	3	—	1	—	2

(*b*) *Foreign body not removed* (Table III*b*).—10 intra-ocular foreign bodies were not removed. 4 of these, 2 air-gun pellets and 2 fragments of cartridge, were non-magnetic, and the eyes were lost. Of the remaining 6, 2 were shown to be magnetic after enucleation, and the history makes it probable that the others were also magnetic, though 2 are retained without evidence of siderosis.

Uveal trauma.—It is generally held that damage to the uvea, particularly to the ciliary body, increases the seriousness of a perforating injury.

(*a*) *Iris damage* (Table IV).—The cases with purely anterior-segment wounds, in which traumatic cataract did not complicate the picture, have been analysed to show the effect of iris injury. In Table IV

TABLE IV.—INFLUENCE OF IRIS DAMAGE ON RESULTS
ANTERIOR CHAMBER INJURIES WITHOUT TRAUMATIC CATARACT: 64 CASES

	No. of cases	Result known	Excised	6/9 or better	6/12 to 6/24	6/36 to 4/60	<4/60	Result unknown
Iris undamaged	18	14	—	9	3	1	1	4
Minor iris damage	18	17	1	10	2	1	3	1
Major iris damage	28	17	3	9	3	—	2	11

minor iris damage includes nicks and small holes in the iris, and fine anterior synechiæ; excision of iris prolapse, or an iridectomy necessary at operation constitutes major iris damage. The results are known in 48 cases, and suggest that iris damage had little influence on visual results, though it did perhaps involve a greater risk of loss of the eye.

(*b*) *Anterior synechiæ.*—Table V shows the results in 26 cases where anterior synechiæ were present.

TABLE V.—RESULTS OF ANTERIOR SYNECHIÆ: 26 CASES

	No. of cases	Result known	Excised	6/9 or better	6/12 to 6/24	6/36 to 4/60	<4/60	Result unknown
Divided	10	10	2	6	1	1	—	—
Not divided	16	13	5	1	4	—	3	3

Those classified as not divided include patients where division was attempted unsuccessfully or with only partial success. The results are considerably better when the synechiæ were divided, whether traumatic cataract was present or not. Though successful division is easier in an eye which is already doing well, the results bear out the accepted teaching that these synechiæ are potentially harmful.

(*c*) *Wounds of the ciliary region* have also been studied, as, by tradition, they are particularly serious. As I wished to exclude limbal wounds which had involved damage to the anterior segment only, the following were taken as criteria of what constituted a wound of the ciliary region:

(1) Perforating wounds of the sclera within 3 mm. of the limbus.

(2) Corneoscleral wounds extending well across, or well round the limbus.

(3) Limbal wounds through which an intra-ocular foreign body had entered and been removed from the posterior segment.

(4) Limbal wounds with iris prolapse in which later examination through the coloboma showed scarring in the ciliary body, or zonular region.

There were 25 cases in all, giving the results shown in Table VI. As one would expect, when the

TABLE VI.—RESULTS OF WOUNDS OF CILIARY REGION: 25 CASES

	No. of cases	Result known	Excised	6/9 or better	6/12 to 6/24	6/36 to 4/60	<4/60	Result unknown
Without traumatic cataract ..	13	11	2	7	1	—	1	2
With traumatic cataract ..	12	11	9	—	—	1	1	1

wound was complicated by traumatic cataract, the results are uniformly bad. It is, however, surprising to find that when the lens was clear these wounds gave a low excision rate, and that 7 out of 13 cases got vision of 6/9 or better. I am fully aware that these numbers are too small to prove anything, but they suggest that perhaps the outlook in these wounds is less gloomy than was supposed, provided the lens is undamaged.

Traumatic cataract.—It is clear that the presence or absence of traumatic cataract is the most important single factor in the prognosis of perforating injuries. Table VII gives the results of the

TABLE VII.—RESULTS IN CASES WITH TRAUMATIC CATARACT: 46 CASES

	No. of cases	Result known	Excised	6/9 or better	6/12 to 6/24	6/36 to 4/60	<4/60	Result unknown
Without intra-ocular foreign body	24	23	11	3	—	2	7	1
With intra-ocular foreign body	22	18	12	3	—	—	3	4
Total	46	41	23	6	—	2	10	5

46 cases in which an attempt was made to save the eye. 23 eyes were lost, a rate of 50%, against 14% in injuries where no traumatic cataract was present. The recorded vision in these aphakic eyes is the best obtained with a correcting glass, regardless of whether the patient was using the eye. Of the 41 known results, only 6 had vision above 6/36. Table VII also shows that the presence or absence of a removable intra-ocular foreign body did not materially affect the issue.

Age is likely to have a considerable influence on the behaviour of a traumatic cataract, and Table VIII shows the results according to age. If one divides the cases into those under and over 31, it is

TABLE VIII.—TRAUMATIC CATARACT—RESULTS ACCORDING TO AGE

Age	Total	Excised	6/9 or better	6/12 to 6/24	6/36 to 4/60	<4/60	Result unknown
0–10 ..	12	5	1	—	1	5	—
11–20	5 (23)	2 (9)	aged 10 (3)	—	— (2)	1 (6)	1
21–30 ..	6	2	1	—	1	—	2
31–40 ..	11	6	2	—	—	1	2
41–50 ..	4 (23)	3 (14)	— (3)	—	—	1 (4)	—
51–60 ..	3	1	1	—	—	1	—
Over 61	5	4	—	—	—	1	—

apparent that more eyes were lost in the older age group, but the number achieving good vision is the same in both groups. Results in the 0–10 group suggest that in childhood more eyes are saved but amblyopia often prevents useful vision.

This gives a gloomy picture of the results of traumatic cataract and one recalls the old dictum that traumatic cataract in a child means the loss of the lens, and in an adult the loss of the eye. In this series the loss of the lens in a young child has also meant the loss of useful vision. Contact glasses were not used in enough cases to comment on their value in traumatic aphakia.

Localized lens opacity.—I was surprised to find in this series 17 patients with a localized lens opacity (Table IX). 15 have been observed more than three and a half years after the injury, 3 for as long as

TABLE IX.—RESULTS OF LOCALIZED LENS OPACITY: 17 CASES

Result known	Excised	6/9 or better	6/12 to 6/24	6/36 to 4/60	<4/60	Result unknown
15	—	9	5	1	—	2

seven years. In 7 cases the lens damage was noticed on admission, 5 being associated with a foreign body in the lens or in the posterior segment, and 1 more with a definite wound of the lens capsule. In 10 the opacity was noticed later, or only found at the follow-up examination, and it is not possible to be sure when it developed. Those opacities which were found at follow-up examination do not differ in appearance from those known to have been present for years, and in no case has there been significant visual deterioration.

One ill-defined but localized opacity on the posterior surface might have been due to lens concussion, but in the others there is a definite opacity in relation to the original wound. If anterior, this consists of a subcapsular opacity, with either an imprint in the deeper layers of the lens, or localized opacity extending through the cortex. Where the injury has been at the equator the opacity reaches round in both anterior and posterior cortex, and is sometimes sector-shaped. These lens opacities are not surrounded by a zone containing water clefts or lamellar separation, and look stationary. The youngest patient to have a localized opacity was 4 and the oldest 50 at the time of injury. The opacity is consistent with good vision, 9 patients out of 15 having 6/9 or better, and 5 more 6/12–6/24.

The possibility of damage remaining localized is important when dealing with cases where lens injury is observed, but frank traumatic cataract is not present.

SUMMARY

In this series infection has not been a common cause of loss of the eye and is unlikely to be entirely eliminated from a group of perforating injuries. Most eyes were excised for an inflammatory reaction, one of our fundamental problems. The results of wounds caused by magnetic intra-ocular foreign bodies have not differed significantly from other similar wounds if the foreign body could be removed. Cases in which anterior synechiæ were divided have done better than those in which they were not divided, but otherwise the presence or absence of iris damage does not seem to have had an important effect on results.

What stands out most clearly from this investigation is the truth of the recognized teaching as to the seriousness of traumatic cataract. In each group of cases the results have been made much worse by its presence. The age of the patient has made little difference to the visual loss.

Two findings are rather unexpected, and it would be rash to draw conclusions without confirmation from much larger numbers.

(1) Wounds of the ciliary region have given relatively good results when traumatic cataract was not present.

(2) 10% of patients had, in relation to their wound, a localized lens opacity which had in some cases remained stationary for as long as seven years. These opacities did not prevent a good result, and carried a much better prognosis than traumatic cataract.

The question arises whether these opacities are as common as this series suggests, and whether, in fact, they remain stationary indefinitely. They indicate that in dealing with injuries, every care should be taken not to extend local lens damage.

Miss Savory, in reply to a question by **Professor A. Sorsby** as to whether she could say what happened in the category of limbal wound cases uncomplicated by other serious injury, said that she had not analysed her cases from that angle. Most of these patients were suffering from complicated wounds, but some, with traumatic cataract, had quite small penetrating wounds caused by a foreign body which was removed via the wound of entrance. These did badly.

Mr. F. A. Juler said that the statistics of this subject opened up many points of interest. One point which Miss Savory had raised was the way in which traumatic cataract often resulted in such poor permanent vision. Some time ago (1921, *Trans. ophthal. Soc. U.K.*, **41**, 129) he had carried out some work on this subject among small children and he had had a number of cases in which the time of injury was established. It seemed that if the child's traumatic cataract formed before the age of 5 he would always have poor central vision (6/36 or less), but if after the age of 7 he would get 6/12 or better. This might throw some light on Miss Savory's figures.

Mr. Humphrey Neame thought it likely that the better prognosis in wounds over the ciliary region in this series might be explained by the introduction of needles and sutures, which were used to close the scleral wound, leading to quicker and cleaner healing.

The President had at one time collected a series of intra-ocular foreign bodies and had arranged them according to weight. He wondered whether Miss Savory could tell him the relation, which might be an important one, between the final result of the cases and the weight of the foreign body.

Miss Savory replied that very often the exact size was not given. Sometimes the notes stated that a large foreign body had been removed, and it appeared that, in general, the bigger the foreign body the worse the case was likely to do. It was unfortunate that exact sizes were not recorded on the notes as a routine, either for the wound or the foreign body.

Mr. Affleck Greeves said that when the foreign body was of considerable size retinitis proliferans would be more likely to follow.

Miss Savory said that there were 5 cases of retinitis proliferans in her series. One of these was a man with a large wound in the upper part of the sclera and another a child with a large limbal wound. 3 cases had had intra-ocular foreign bodies, none very large, but all situated far back on the retina.

Section of the History of Medicine

President—

The Right Hon. Lord WEBB-JOHNSON, K.C.V.O., C.B.E., D.S.O., T.D., M.B., F.R.C.S., LL.D.

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Two Reformers in the Army Medical Services

By Lieut.-Colonel J. B. NEAL, T.D., M.R.C.S., L.R.C.P.

I.—SIR WILLIAM MUIR

IN 1843, Sir James McGrigor had been Director-General of the Army Medical Department for twenty-eight years, Sir John Webb, Director-General of the Ordnance Medical Department for thirty years, and George Renny, Director-General of the Irish Medical Board for forty-eight years. This was no time for revolutionary change, and indeed, the organization of the medical services of the army had not altered in principle since Britain had had a standing army. But changes were to be effected in the two following generations, and William Mure Muir, in 1843 an assistant surgeon of one year's service, and William Taylor, born on April 5 of that year, were to contribute largely to them. The foundation stone of the system from 1660 until 1873 was the regimental hospital run by a regimental surgeon, appointed either by purchase or by patronage, as Muir was, or, after the Crimea, by competitive examination. The regimental hospital was as integral a part of the regiment as its officers' mess or quartermaster's stores. It was staffed by a surgeon and his assistant surgeons, distinguished in dress from other officers of the unit only by the instrument case and a plume of black cock's feathers in the head-dress, who were at once the physicians, surgeons, pathologists and, somewhat unofficially, accoucheurs to the regiment which was their practice and their world. By and large they relied for the rest of their lives upon the knowledge with which they entered the service, for foreign tours were long—twenty or thirty years was not uncommon—and the taking of leave involved the officer in responsibility for paying a substitute. The change to the present system of general hospitals staffed by specialists, was achieved by stages, with the two most important of which Muir and Taylor were intimately concerned.

Muir graduated M.D. in Edinburgh in 1840 and was commissioned as assistant surgeon in the 42nd Foot (now the 1st Black Watch) in 1842. He was promoted surgeon into the 33rd Foot (now the 1st Duke of Wellington's Regiment) in 1854, served with them throughout the Crimean campaign, and became Deputy Inspector-General in 1858. As such he went to China in 1860 as Principal Medical Officer of Sir Hope Grant's expedition, being specially promoted to Inspector-General, appointed C.B., and mentioned in despatches. The campaign was medically notable for two reasons: it was the first in which an officer was appointed specifically for sanitary duties, and Muir's report appears to be the earliest record of his views on the regimental system. To anticipate for a moment, Lord Wolseley, who held a high opinion of Muir, referring to him as "one of the very ablest men in our army", held Sanitary Officers in very low esteem. In his book, "The Soldier's Pocket Book for Field Service," he writes: "The Sanitary Officer is a creation of recent years, and as a rule he is a very useless functionary. In the numerous campaigns in which I have served with a sanitary officer, I can conscientiously state that I have never known him make any useful suggestion, whereas I have known him to make many silly ones."

Muir established general hospitals in China, and in his report he notes "the great superiority of the general hospital system as a whole over the pure or modified regimental system, and the total incapability of the latter singly adapting itself to the requirements of an army in the field".

"The testimony of Jackson, McGrigor, and other surgeons of large experience in the field, in favour of the regimental system, is entitled to great weight, although it must be recollected that this opinion was founded on experience of evils in the general hospital system now known not to be necessarily inherent in it, and to a great extent preventable. Moreover, the regimental system is so essentially of the British Army, that it will probably hold its ground for some time, both in war and peace, but must eventually give way to the more comprehensive and practical general hospital system, as this last must always take precedence in time of war, with a view to which all peace establishments must be modelled."

"In the meantime, a medical officer in charge of an army in the field would have to work both systems. . . ."

As regards the testimony of other surgeons, John Hunter's death has been attributed to a

difference of opinion with a colleague upon the rival merits of general and regimental hospitals, though a dispute with John Gunning (the uncle) over fees for surgical lectures has also been blamed.

Muir's practical mind is illustrated by the last recommendation in the China report: "That the present scale of supplies, stores, &c., for the sick of an army on field service might be advantageously reduced. For instance, garden benches can always be made anywhere, and take up too much freight; nor is it necessary to have a chamber-pot and pudding-shape for every man of the force."

From China, in 1861, Muir went to Canada as Principal Medical Officer of the force sent there as a precautionary measure in consequence of the "Trent" incident, but he crossed the border into the United States, not as P.M.O. of an invading army, but as a liaison officer. He accompanied Sherman on his march "from Atlanta to the sea", and in his report stressed Sherman's action, before he set out, in "disencumbering the force of its sick and wounded". In point of fact, Muir's completing the march is a tribute to his stamina, for despite its size, Sherman's force was a hand-picked body of men, and an American account speaks of his moving "at a pace incredible to anyone who did not know he had an army of pioneer backwoodsmen and hardly believable to those who did".

As P.M.O. in India from 1868–72, Muir tried to introduce general hospitals, but he saw that the time was not ripe. The moral was pointed, however, by the Franco-Prussian War of 1870, and Surgeon-Major Fitz-Gerald, our Commissioner at the headquarters of the German army, reported: "In the field, when a soldier becomes sick, or is disabled by wounds, he ceases—until cured and fit to return to duty—to have any connection with the regiment to which he belongs. . . . Although the sick and wounded are removed from their regiments, they are not sent indiscriminately to the rear of the army to be treated in any general hospital that may happen to be established, as appears to have occurred with the English in the early part of the Peninsular War;" (shades of the "Belem Rangers") "they are admitted and treated in—so to speak intercepted by—properly organized field hospitals. . . ." Innes, Muir's successor in India, had no difficulty in establishing field hospitals for active-service purposes, and Muir, now in the War Office as head of the Sanitary Branch, strengthened the hand of Sir Galbraith Logan, the Director-General, in securing in 1873, the abolition of the regimental system throughout the British Army, except in the Household Cavalry and Foot Guards, and in India. In retrospect, it is difficult to believe that regimental hospitals and regimental surgeons could long have survived Mr. Cardwell's reorganization of the infantry, in 1881, into two-battalion regiments, each with a territorial home and fixed depot, composed of men frequently interchanged between the battalions and, in any case, serving on shorter engagements than had ever previously been known in the British Army.

In 1874, Muir succeeded Logan as Director-General of the Army Medical Department, and the slow process of consolidation began. From the start, Muir had to face not only criticism from without, but also dissatisfaction among the officers of the Department. The abolition of the regimental system was not popular, and there was a strong feeling that the Government had broken faith with the medical officers. The creation of a united corps of medical officers achieved something for which Muir had worked and preached for many years, and he felt that until time had healed the wounded feelings of the surgeons uprooted from their regimental homes, more could be gained by turning his attention to the other end of the scale by bringing the Army Hospital Corps further into the medical orbit. In 1875, direct enlistment was permitted to the Army Hospital Corps, hitherto filled entirely by transfers; the Depot was moved from Netley to Aldershot and—a great stride towards autonomy—a medical officer, Deputy Surgeon-General W. A. Mackinnon, was appointed to command it, with another, Surgeon-Major Sandford Moore, as chief instructor. In 1877, the powers of command of medical officers were for the first time defined, not, indeed, to everyone's liking, but still, something had been won. The records of the A.H.C. were transferred from the Purveyors' Department of the War Office to the D.G., A.M.D., and, in 1882, for the first time, a medical officer, Surgeon-Major William Johnston, was appointed to Army Headquarters as Staff Officer for the A.H.C. Muir, typically Scots, combined firmness of purpose with caution of approach, and these advances, small in themselves, were welding the new Department together and preparing the way for the issue of the Corps Warrant in 1898.

It will be recalled, from my quotation from Muir's report on the China expedition, that his objection to the regimental system was based upon its unsuitability for war. The move of the Depot to Aldershot in 1875 brought the Army Hospital Corps into intimate contact with a large body of fighting troops constantly engaged in field training, and there was thus a magnificent opportunity to develop a war organization under the new system. The Prussian model was taken as a foundation, and upon this was built the edifice which has continued, although with modifications, to the present day. A medical officer was attached to each battalion or equivalent unit, which provided stretcher-bearers from its own ranks. A Bearer Company had the responsibility of clearing the wounded from the battlefield and conveying them to Field Hospitals. The analogy with the modern Field Ambulance and Field Dressing Station is close, and then, as now,

once a wounded man passed beyond the care of his Regimental Medical Officer, his unit ceased to have any responsibility for him. Luckily, there was time for this method to be devised, taught and assimilated, for it was not required in actual battle until 1879, when colonial operations in South Africa allowed it to be tested under the stress of war.

But we must turn back to 1878. Muir knew, and it is clear to-day, that he was doing what was right, but it was less apparent to his subordinates. Dissatisfaction was intense: medical officers were leaving the service and, at each entrance examination, candidates were fewer than the declared number of vacancies. A departmental committee, with the Secretary of State for War as chairman and Muir as a member, was set up to consider the grievances and to suggest remedies. Some of the grievances have a strikingly modern ring—that pay did not keep pace with incomes obtainable in civilian life, loss of allowances, difficulty in obtaining quarters, uncertainty of tenure of appointments, slowness of promotion. For some, explanations had to suffice; for others, remedies were proposed, and some of these were accepted by the Government. In particular, the "10 years' system"—what we should to-day call Short Service Commissions—was modified. But one of the grievances alleged was the abolition of the regimental system, and of this the committee reports—and the answer smacks of Muir—"We have made these observations in the hope of reconciling medical officers to the unified staff system; but not with any idea of discussing the relative advantages of this and of the regimental systems. We look upon that question as having passed beyond the stage of argument."

It is probably true to say that, to the end, Muir was respected rather than liked, and the opposition which he encountered prevented his adding more than small bricks to the unified structure before his retirement on May 7, 1882, but the way was now open for further advances. In January 1885 he was elected Principal of Edinburgh University, but he died less than six months later. His predecessor as Principal, commending him at his election, described him as having "a scholarly mind, a high sense of duty, great powers of application to business, firmness of purpose . . ."; it is not hard to see why, among surgeons brought up to the circumscribed but easy-going life of regimental duty, the chief apostle of the change to the more disciplined ways of service in a department as yet lacking tradition should have inspired esteem rather than affection. But it was a lasting esteem, for thirty years later, Sir Alfred Keogh could say: "See to it, gentlemen, that these young officers are brought up in the principles of McGrigor and Muir."

II.—SIR WILLIAM TAYLOR

In Part I of this Address, I considered the work of Sir William Muir in replacing the regimental hospitals of the British Army, run by regimental surgeons, by general hospitals with a complement of officers of the Army Medical Staff and other ranks of the Army Hospital Corps. Between Muir's retirement in 1882 and Taylor's appointment as Director-General in 1901, a great deal of development took place. In 1898, officers and other ranks were united in one corps, the Royal Army Medical Corps, which included for a time even the medical officers of the Household Brigade. Officers up to and including Colonels were borne on the Corps list, with the simple titles of military rank which we now use, the prefix Surgeon remaining in use only for General Officers, who were still styled Surgeon-General. Officers of the Corps had full powers of military command over its other ranks.

But there were as yet no specialists in the Corps. Study-leave was still unknown. There was no co-operation with the civilian side of the profession. Equipment was a mixed bag, for although radiological equipment was in use in the Egyptian Expeditionary Force in 1898, the microscopes in the Army Medical School (now the Royal Army Medical College) dated back to 1860. Many of the advances in these directions were due to the work of Sir William Taylor.

A generation younger than Muir, Taylor was born in 1843, graduated in Glasgow, and entered the Army Medical School at Netley in 1864. The School, one of the post-Crimean Herbert-Nightingale reforms, had been opened at Fort Pitt, Chatham, in 1860 to give young officers a grounding in the military aspects of their professional work. The subjects of instruction were military medicine and surgery, hygiene and pathology. Entry was by competitive examination, and a passing-out examination was held at the end of the course, the combined marks determining the order of merit, and hence the seniority of the "Medical Candidates", as they were called.

Taylor passed in 26th out of 77, lower than he might otherwise have been owing to his obtaining only 25 marks out of 300 in Natural History. He passed out 19th—an average man of an average batch—and was commissioned Staff Assistant Surgeon. He went almost at once to Canada, took part in quelling the Fenian Rising, and gained experience of the regimental system by his appointment in 1870 to the Royal Artillery. He had a long spell of Indian service, including two campaigns on the frontier and two in Burma, and four years as Surgeon to the Commander-in-Chief. Taylor then went farther East, as liaison officer with the Japanese forces in the Sino-Japanese war of 1894-5.

In 1895, as a Surgeon-Colonel of one year's standing, he was appointed Principal Medical Officer of the Ashanti expedition, where his rules of health, except for that which enjoined

the constant wearing of a helmet by day and of a flannel belt by night, would have passed current in any tropical theatre in the late war. His reward was accelerated promotion to Surgeon-Major-General.

In 1898, he was P.M.O. in the Egyptian campaign, the R.A.M.C.'s baptism of fire, and in the following year, he became P.M.O. of Her Majesty's Forces in India. This was certainly the turning-point of his career. He was virtually an independent authority, and able to put into effect his views upon health and, perhaps more important, he was denied any part in the South African war.

As I have mentioned, there were no specialists in the Corps until Taylor introduced them in India. The beginnings were small—four Specialist Sanitary Officers for the whole of India and Burma—but this is the important point, each bore the personal designation of "Specialist in the Prevention of Disease". This advance was followed by the appointment of officers to hospitals with some security of tenure, for specialized professional duty as physicians and surgeons, though the appointments carried no extra pay and they lapsed when the officer left India.

But Taylor was by no means one for all work and no play. It was deemed an eccentricity in Leishman that when he first went to India he took a microscope. There is no record of the contents of Taylor's baggage, but it may well have contained a saddle, for he first whipped in to, and was later Master of, the Peshawar Vale Hounds.

Meanwhile the South African war was being fought, criticism was directed at the medical services, the Royal Commission on the War in South Africa deliberated, and in 1901 a new Director-General had to be found. Mr. Broderick, Secretary of State for War, set up a committee to consider the reorganization of the Army Medical Services, and the incoming Director-General would have to give effect to its recommendations. The existence of the appointment itself was for a time in the balance. There was talk of abolishing the post completely, and replacing the Director-General by a board of officers to act as advisers to the Adjutant-General. The infusion of new blood by the introduction of an eminent civilian was suggested, and the name of Sir Frederick Treves was canvassed for the appointment. Meanwhile, there was an interregnum and, for the first time, an officiating Director-General, Surgeon-General A. F. Preston, was nominated to administer the Department while a decision was being taken. The choice was made, and the lot fell upon Taylor. He had shown notable administrative ability, he was deeply concerned for the health of the soldier and for the professional standards of the Corps, and he was completely untainted by any South African connexions. He assumed office on December 2, 1901.

Most of the Broderick Committee's recommendations were put into effect, though not without opposition, both overt and disguised as help. The new Director-General was made largely dependent upon the "advice" of the newly constituted Army Medical Advisory Board, composed of a large and eminent civilian element and a somewhat embarrassingly constituted military one, and in the end the tension told upon Taylor and led to his resignation.

Much of the task set him by the Broderick Committee was close to his heart, especially the introduction of specialist appointments, with specialist pay, and the opportunity for professional study at the expense of the Crown. Officers on entry to the Corps might be granted seniority up to twelve months as credit for experience in approved appointments held in civil hospitals before joining. Every officer in his qualifying examination for promotion to major was required to offer a special subject, and among those which might be offered were dental surgery, pædiatrics, psychological medicine and skiagraphy.

But whereas the qualifications for promotion, the rules for the conduct of examinations and the conditions of specialist training were laid down in strict detail, and closely watched by the Advisory Board, the last recommendation was left in general terms: that "a Military Hospital and Medical Staff College for training of officers would add materially to efficiency, and immediate steps be taken by the Advisory Board to prepare a scheme for the establishment of one". To this scheme, Taylor whole-heartedly devoted himself.

The Army Medical School was transferred from Netley to hired premises in London, in order to take full advantage of the teaching facilities of the metropolitan hospitals, while the Royal Army Medical College and Queen Alexandra's Military Hospital, exactly as we now know them, were being built. Taylor's interest in professional training had become known, and the presence on the Advisory Board of Alfred Fripp, Frederick Treves and Cooper Perry opened the doors of London's hospitals to the keen and able army medical officer. Many of the teaching hospitals had on their staffs men who had served in South Africa, either with the so-called "Civilian War Hospitals" or as consultants (with the honorary local rank of Lieutenant-General and pay of £5,000 a year), and they had a recent picture of the needs of the military surgeon. Taylor was quick to seize these opportunities of turning suspicion and even enmity between the civil and military branches of the profession into co-operation. Taylor's work for specialization and training in professional subjects, and for ensuring proper standards of qualification for advancement, stands to this day; modified, as we think, for the better, but that was inevitable—he was in office for only three years—and he would have had it so.

JOINT MEETING No. 2

Section of Surgery with Section of Anæsthetics

Chairman—Professor F. A. R. STAMMERS, C.B.E., T.D., Ch.M., F.R.C.S.
(President of the Section of Surgery)

[*February* 4, 1953]

DISCUSSION ON HYPOTENSION DURING ANÆSTHESIA

Dr. J. B. Wyman: "We have reason to hope that the day will come when hæmorrhage will be controlled by a quicker procedure than the awkward, time-consuming ligature." These words were said by William Stewart Halsted (Blalock, 1952) almost fifty years ago and very little progress had been made in this direction until Griffiths and Gillies (1948) introduced a high spinal technique for deliberately inducing hypotension in order to procure a bloodless field at the site of operation.

There are many ways of achieving hypotension, but the introduction by Paton and Zaimis (1948) and Barlow and Ing (1948) of the lower members of the methonium series, which specifically blocked the autonomic ganglia, led Davison (1950), Enderby (1950), Scurr (Organe, 1950) and other workers to use a relatively simple procedure for lowering the blood pressure. They stressed its value to the surgeon and the question arises: Is such a drastic procedure justified merely as an aid to surgical technique? But this may not be the only function of the drug. By virtue of its ability to block the autonomic ganglia it may be able to protect the patient not only from shock due to blood loss but also from shock due to trauma. It may also, by its ability to block the autonomic ganglia and thus produce vasodilatation, prevent established shock from becoming irreversible.

Its future in surgery may not merely be limited to the production of a so-called bloodless field, but also as a means of preventing and controlling shock in major surgery.

I shall discuss the results of our experience in 1,000 major surgical cases that have been anæsthetized by this technique.

It is, of course, important to differentiate between the sequelæ caused by the hypotension and those that ordinarily follow anæsthetic and surgical procedures.

Soon after we began to use C5 it became evident that although we could not always produce an absolutely dry field we could always diminish the blood loss considerably. In the beginning we limited the cases to those in which the blood loss was excessive or where it impeded the surgeon. Three things were noted immediately: (1) The excellence of the working conditions provided for the surgeon. (2) The evenness of the patient's general condition during the operation. (3) The remarkably fine condition of the patient following the operation.

This may have been due merely to the conservation of blood, but when we extended the technique to those cases where the blood loss was considered of no importance we found that they, too, did remarkably well. It appeared to us that these patients were protected from trauma as well as from the effects of blood loss. It is for this reason that this series contains a large number of cases where its use may be considered unnecessary on the basis of limiting the blood loss or in the production of a dry field.

It has been demonstrated by Freeman *et al.* (1938) that in a controlled series of dogs, in which an attempt is made to induce shock by hæmorrhage, those that had been sympathectomized never reached the stage of irreversible shock. When these animals were sacrificed there were none of the histological changes in the liver, kidneys, and intestine that normally accompany irreversible shock. Paton and Zaimis (1951) have shown that pentamethonium iodide inhibited the effects of stimulation of the peripheral stump of the vagus cut in the neck.

In addition to this animal work there is a certain amount of clinical evidence to support this theory that paralysis of the autonomic ganglia may protect the patient against shock. Operations which formerly gave rise to shock and great anxiety can now be done under light general anæsthesia and C5. The general condition of these patients on the same day is excellent.

We have had many devastating criticisms and objections to the use of this drug: Wilfred Trotter once wrote: "A little self-examination tells us pretty easily how deeply rooted is the fear of the new, and how simple it is when fear is afoot to block the path of the new idea by unbelief and call it scepticism, and by misunderstanding and call it suspended judgment." I once had the greatest difficulty in persuading my colleagues that the renal failure from which a patient died could not be attributed to C5 or C6, if only because the patient had not been given any.

In investigating the use of hypotension in surgery, our concern was (i) the adequacy of the circulation at these low blood pressures to the major organs, (ii) the possibility of the occurrence of vascular accidents. The technique was used in 1,000 major surgical cases embracing most age-groups and most types of operations.

Contra-indications

(i) Asphyxia. (ii) Severe heart lesions. (iii) Severe renal damage. (iv) Established shock. (v) Established hæmorrhage. Some of these may yet prove to be false, whilst others may have to be added: experience alone will help us to decide.

Complications

I. *The Brain:*

In this series we have had one case of fatal cerebral thrombosis. This followed a Smithwick's operation on a man who had been refused operation a year previously on the grounds that his disease was too far advanced.

There has also been one case in which the patient failed to recover consciousness. This patient had a two-stage operation. I anæsthetized the first stage, using the hypotensive technique and the patient made a perfectly straightforward recovery. The second stage was attempted later and, unaccountably, the precautions we had decided upon were not taken. The patient was not intubated, the anæsthetist was working on his own, and there was no ready means of intravenous therapy. During the operation control was lost of the airway and there was a period of prolonged asphyxia. I feel that in this case there was a serious error in technique and it was an avoidable catastrophe. It once more demonstrated that hypotension and anoxia are incompatible.

During this time two other cases, both undergoing Smithwick's operation and both having had previous cerebral mishaps with residual neurological signs, appeared worse mentally after the operation. Psychical disturbance in this type of patient is not unknown after operations.

We have used the hypotensive technique for cranial surgery with excellent results. The condition provided for the surgeon may influence the scope of surgery in this field. The bloodless scalp and the flaccid brain cut down the operating time by hours.

II. *The Heart:*

When we first commenced to use this technique we found that the ECG readings showed no change during the period of hypotension or post-operatively, but we had not taken a sufficient number of heart leads. We now find that there is a lowering of the T wave or a depression of the S-T interval in some of these leads, indicating a certain amount of cardiac ischæmia. The depression is not great, it is reversible, but it may persist post-operatively for a few days.

We have had one case of cardiac arrest early on during this series and it was from this that we formed the basis of our contra-indications and precautions. This case illustrated the dangers of giving large doses of Pentothal to asphyxiated patients. The heart was restarted with cardiac massage but the patient died two days later.

Towards the end of this series the same mistake was reported by a junior member of our staff who got into trouble during induction and who had not known of the precautions we had laid down. An elderly lady of 77 was given C5 after a period of asphyxia during induction, and the heart stopped. This was restarted, but the patient failed to regain consciousness and died later. The post-mortem revealed gross atheromatous changes in all the vessels, and it was suggested by the pathologist that this was a case of coronary thrombosis. This, however, was not demonstrated to my satisfaction and I think this avoidable death was due to inducing hypotension in a patient with a myocardium already affected by anoxia.

Of these thousand cases, no fewer than 114 had an initial blood pressure of over 200 mm.Hg. In 217 the blood pressure was between 160 and 200, and in 38 cases under 100 mm.Hg. 11 patients were fibrillating, several cases had previous coronary disease, including one in heart failure at time of operation. From my observations on these cases with cardiac lesions it appeared that these did better than with the ordinary anæsthetic. The heart working against a lower load appeared to beat away unruffled by the trauma and by any fluctuations in circulatory volume.

It is typical of these C5 cases that once under control the blood pressure and pulse-rate remain very steady. The peaks and depressions that come with other anæsthetics do not appear, and the results are reflected in the post-operative condition.

The incidence of post-operative hæmatomata went down as the surgeons became accustomed to the technique. This incidence was much lower in the first 100 thyroidectomies done under C5 than in the previous 100 done without. Once the surgeon grasps the idea that vessels that appear to be bleeding only slightly must be tied, there is no reason to fear post-operative hæmorrhage. The bleeding points can so easily be seen, and hæmostasis is more assured. In prostatectomies not only is the hæmorrhage considerably less at the time of operation but also the urine post-operatively becomes clear more quickly and there is less tendency to clot retention. In this series, of 71 prostatectomies there has been one case of clot retention.

III. *The Kidneys:*

There has been one death from renal failure two weeks after operation. The patient had Ellis type I kidneys and each was smaller in size than the adrenals. Such cases were invariably fatal even before the days of C5. Another patient had less than 45% kidney function and although we gave no Methedrine the blood pressure returned on its own, but took a little longer than normal.

There have been over one hundred operations on the urinary tract, including 71 prostatectomies, 11 nephrectomies, 10 transplants of ureters, 4 total cystectomies, 7 nephrolithotomies, and several other explorations and plastic repairs of kidneys. In no case did the post-operative renal function worry us. In some cases, however, we have met with a certain amount of oliguria, but this has been transient only, although on three occasions it exceeded twenty-four hours.

IV. *The Liver:*

There has been no disturbance of liver function as far as we can tell, but one case presented with cirrhosis of the liver three months after thyroidectomy.

The connexion between thyroids and cirrhosis of the liver is recognized, and as far as I can make out this type of cirrhosis takes more than six months to develop.

Discussion

Of these 5 deaths, 3 were due to faulty technique and 2 to the patient's disease. These cases emphasize the great care required in this method and the need for the anæsthetist to understand the fundamental principles upon which it rests. This technique demands a high standard from the anæsthetist and if the basic requirements are complied with then all is well, but if they are not observed the penalties are very heavy.

Many people regard hypotension anæsthesia as an unphysiological procedure but then gastrectomy is an equally unphysiological procedure.

It is the normal physiological response to trauma or hæmorrhage to maintain the cerebral circulation at the expense of the other vital organs.

We endeavour to treat hypotension and shock by transfusions and by vaso-pressor drugs. We now know that the hypotension of itself is not incompatible with life as are the associated conditions and though we cannot otherwise keep a patient alive for long with a systolic blood pressure below 60 mm.Hg, we can with C5. Thus those shocked patients who do not respond to transfusions may be helped by using C5. In this series I have had two such cases. They were both cases of hæmatemesis which transfusion failed to resuscitate, and in whom it was considered necessary to do an emergency gastrectomy. The first was a lady of 67 who had a severe hæmatemesis and melæna. It was decided to perform an emergency gastrectomy and I was presented with a woman with a systolic blood pressure of 40 mm.Hg. She was green in colour, sweating, vomiting blood, and almost moribund. After induction of anæsthesia the blood pressure dropped to 30 mm.Hg. Blood was pouring into the patient but appeared to have no effect. I have had a fair experience during the war of massive hæmorrhage and of shocked patients and I felt that I could not keep this woman alive much longer. I then decided to give her C5, knowing it would make little difference to her B.P. but, at the same time, would alter the distribution of the circulating blood, perhaps relieve the constriction of the vessels to her kidneys and perhaps prevent irreversible shock. A dramatic change took place. The face which formerly appeared to have no circulation at all became flushed and in a short time the patient became warm and dry and there was scarcely any alteration in the blood pressure. She was now, in my opinion, an ordinary case of controlled hypotension and I felt happier about the eventual result. The gastrectomy proved difficult but all appeared to be well even though over an hour had elapsed since commencing the anæsthetic. At that moment the surgeon, whilst reflecting the stomach, pulled on it and the patient's heart stopped. The patient died from what must have been vagal inhibition. I felt later that I had made a mistake in technique. I have already mentioned that C5 blocks the vagus. I have repeatedly stimulated the vagus in cats in order to obtain the slowing of the heart and fall in blood pressure that normally occurs, but have always failed. My error was, I think, in giving a very small dose of C5 (30 mg.), which had worn off after an hour and a quarter. The next time a similar case occurred I gave a much bigger dose. This was an old gentleman of 80 who, tiring of vomiting blood, went into a public house for a pint and promptly perforated. He was extremely shocked and failed to improve after many pints of blood had been poured into him. After induction I could not detect any blood pressure at all and this time I gave 100 mg. as the initial dose of C5. Again there was the dramatic change in the patient's general condition. On rubbing his face it immediately flushed, whereas formerly it had been deathly white, and I must admit to hoping at the time that the same process was taking place in the kidneys and in the brain and in the rest of the patient. The gastrectomy was completed without incident and a fortnight later the patient left hospital for convalescence.

I do not wish to infer from these two cases alone that this is the ideal method of treating shock but merely to suggest a line of treatment in those cases where our present means of resuscitation fail. I do wish, however, to suggest from my experience of this technique that the future of this drug may lie as much in the way of preventing shock during major surgery as in the provision of an ischæmic field at the site of operation.

The Post-operative Course

All patients are sent back to bed on a tilting trolley in the head-down position. This is usually sufficient to raise the blood pressure by at least 20 mm.Hg and the patient is then

nursed in a slight Trendelenburg position for the first few post-operative hours. The blood pressure is taken at regular intervals and usually it fairly quickly climbs back to normal during the next few hours. There are some, however, whose blood pressure fails to return merely by posturing. Methedrine has been suggested as the antidote, but there are several objections to the use of this drug as it is not a true physiological antidote. Phenyl-ephrine (neo-synephrine) overcomes some of the objections as it is not a cerebral stimulant, but the other objections still apply.

Some cases of failure of recovery of the blood pressure are due to dehydration and two pints of saline intravenously will speed things up. I have seen this cause diuresis in a patient with persistent hypotension, dilated pupils, and with oliguria, due to C5. The blood pressure starts going up and the pupils contract. C5 is secreted by the kidneys and the dehydration may start a vicious circle of events as failure to excrete prevents the blood pressure from rising.

It is interesting to note that while the blood pressure remains low the condition of the patients does not give rise to anxiety. They are warm, dry and alert and morphia may be given quite safely even though the blood pressure may still be very low.

These difficulties may be overcome by the use of a new drug which has just been introduced into this country by Dr. Magill (1953). Arfonad or Ro 2-2222 is very evanescent in action and thus will enable us to control more exactly the depth and the duration of hypotension. It may overcome many of the disadvantages of C5.

SUMMARY

These first thousand cases anæsthetized with the hypotensive technique have taught us a great deal:

(1) That it diminishes the blood loss considerably. Where the presence of blood obscures the field it grows these will be overcome and the method will not offer any additional risks at operation.

(2) The operative and post-operative course of patients undergoing major surgical procedure is smoothed out.

(3) That this technique must be learnt and understood in addition to the ordinary techniques of anæsthesia. We have made serious errors in technique and in judgment but I think that as our experience grows these will be overcome and the method will not offer any additional risks at operation.

In addition to thanking my colleagues at Westminster Hospital I must especially thank my anæsthetic colleagues. To the surgeons of Westminster Hospital and Mr. Geoffrey Parker at the Woolwich Memorial Hospital I would like to express my grateful thanks.

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Mr. Robert Cox: *Hypotensive Anæsthesia Produced by the Methonium Drugs.*—Until the introduction of deliberate hypotension, a fall in blood pressure was most commonly due to shock or hæmorrhage and this was accompanied by compensatory vasoconstriction with all its potential bad effects. If the hypotension is accompanied by a maintenance of visceral vessel calibre or even by vasodilatation, the factor which the surgeon has learnt to fear is removed. Moreover, if the vessels do not contract and even dilate—and we believe this happens during the use of C5—the dangers of reactionary hæmorrhage are clearly minimized. As a result of the fall in blood pressure induced by C5 the amount of blood lost during an operation is reduced. If a vessel of any moment is cut it bleeds obviously and must be dealt with immediately and a meticulous attention to all bleeding vessels is absolutely vital in using this technique.

The benefits accruing from a bloodless field are increased vision and exposure leading to ease of operation and reduction of operating time without increase of haste. Some operations are made difficult or even impossible by bleeding, notably that of fenestration for otosclerosis.

The operations in which I have used C5 for hæmostatic reasons are radical mastectomy, thyroidectomy and other operations in the neck, prostatectomy, abdomino-perineal resection of the rectum, and thoracotomy done in my hands for lesions in the œsophagus.

Appreciation of the amount of blood lost is apt to be very inaccurate unless measured. We have done this in a number of cases by collecting all blood from sucker apparatus, swabs and soiled operation towels and soaking these in a known quantity of water to which ammonia has been added. A measured quantity of this solution has its hæmoglobin % estimated and comparison with patient's known hæmoglobin % allows an estimate of loss in c.c.

Examples are:

	Without C5	With C5		Without C5	With C5
Thyroidectomy	260 c.c.	30 c.c.	Thoracotomy	1,160 c.c.	160 c.c.
Radical mastectomy	690 c.c.	32 c.c.	Pelvic evisceration (Brunschwig)	1,500 c.c.	160 c.c.
Prostatectomy	550 c.c.	30 c.c.			

It is obvious that savings of this order help both surgeon and patient.

To show the alteration which has come about I have looked up my prostatectomy records since 1949 and reviewed the blood transfusions given during the operation or in the twenty-four hours immediately following. The results are:

No C5: 32 cases with transfusion incidence of 1 case in every 2.

Using C5: 44 cases with transfusion incidence of 1 case in every 6.

Despite this it would be wrong to imagine that serious bleeding cannot occur with C5. In one case I made an error of diagnosis and in trying to enucleate a malignant prostate got into considerable difficulty which was accompanied by furious hæmorrhage which could only be controlled by plugging. The patient lost 3½ pints (in the sucker) in a very short time and Plasmosan had to be used at first for replacement. Despite this the patient looked in fairly good condition at the end of the operation—good volume, pulse just over 80; warm and not sweating, not the picture my experience would lead me to expect in a man in these conditions if not given C5. I shall say more about this aspect later.

Reactionary hæmorrhage is always a hazard in prostatectomy and we have found no increase in its incidence since we have started to use C5. We have had 1 patient out of 44 cases who had to be taken back to the theatre to have his bladder emptied with an evacuator. Prostatectomy is, of course, a short operation and the hypotension need not be of long duration.

In contrast, thoracotomy tends to be a fairly prolonged operation. Using C5 I have done 13 thoracotomies or abdominothoracic operations for œsophageal obstruction, all save one due to malignant disease, and on patients varying in age from 28 to 79. The operations varied in time from 2½ hours to 6½ hours. The 6½ hour operation was on a man of 58 with carcinoma of the stomach involving the lower end of the œsophagus, there being a thoracic stomach with a short œsophagus. The man was very fat with a deep barrel chest and further difficulties arose from the abnormal vascular arrangements of the stomach. At the end of this long and punishing operation, the patient's condition was remarkably good. His blood pressure had been between 60 and 80 throughout most of the procedure. Thirty-six hours later his condition deteriorated suddenly and he died in a few hours. Post-mortem showed massive necrosis of practically the whole stomach and it might be suggested that the prolonged hypotension was a factor in determining the thrombosis of the gastric blood supply, but doubtless the division of many vessels and rough handling during operation were also contributory.

The oldest patient in this series, aged 79, had a carcinoma of the œsophagus. His condition after 4½ hours operating with blood pressure about 60 was remarkably good and remained so for the next two days. Unfortunately, he died on the fifth post-operative day of widespread broncho-pneumonia, an occurrence which does not seem to be attributable to C5.

With regard to other types of surgery: The orthopædic surgeons at Westminster Hospital are quite definite that C5 makes for great improvement in such procedures as laminectomy for prolapsed disc, operations for recurrent dislocation of the shoulder and arthroplasty of the hip joint, in which operation the use of C5 is almost routine.

The plastic surgeon says that he finds its use in mammoplasty enables him to cut his operating time by as much as one hour but thinks that he gets a little more trouble with hæmatomata. He likes to have C5 used in nasal reduction where the bleeding is reduced remarkably.

In operations for hypertension it is being used more and more.

In thoracic surgery, with the incision of access, the advantages of hypotension are obvious but when the operation is on the lung with its low pressure circulation, there seems to be less reason to desire hypotension than in the higher pressure systemic circulation. But in cases of operations on the great vessels its advantages are obvious. In a recent case of resection of coarctation of the aorta the giving of blood was not required until the anastomosis was under way, whereas with conventional anæsthetic methods transfusion is usually started during the opening of the chest. The tendency of aortic clamps to slip is, of course, also reduced.

Disadvantages.—The incidence of reactionary hæmorrhages and hæmatoma formation in my hands is not greater than with normal methods but any relaxation of a meticulous technique of hæmostasis would probably lead to trouble in this direction. Thrombotic complications have also not been a trouble—I have had two deaths from pulmonary embolism, but so small a number of cases as mine is not statistically significant and the incidence does not seem to be higher than with normal methods. Renal failure has not occurred in my cases. Urine continues to be secreted as we have proved by ureteric catheterization of patients with pressures below 80 mm.Hg. Hepatic failure has not been seen.

Once they have become accustomed to the technique, the nursing staff like it.

As a general rule patients are returned from the theatre either in bed or on a tilting trolley

head down. The foot of the bed is kept raised until blood pressure readings, which are taken and recorded half-hourly by the nursing staff, indicate that the pressure is climbing steadily. This usually happens within three hours after return to the ward.

If for any reason it is deemed advisable to accelerate the rise in blood pressure, Methedrine should not be used owing to its central stimulating effect which may deprive the patient of sleep for 24 hours or more after the drug has been given. Neo-synephrine is the drug of choice and it has no unpleasant side effects. In patients with very low blood pressure, oxygen is beneficial. If the patient is restless, this will militate against a return of blood pressure and therefore morphia must be given even with the blood pressure as low as 60 mm.Hg.

The patient is raised slowly by stages and is usually sitting up by the morning after operation. The blood pressure is taken and recorded t.d.s. on the day following operation and this has now in our wards become routine in *all* cases. The record shows some remarkable hypotensions in cases anæsthetized with conventional techniques for standard abdominal operations which would otherwise pass undetected.

On the whole C5 does not make for more work since the extra attention caused by blood pressure readings is offset by not having so much to look after in the way of transfusions and ill, shocked, patients.

The sisters who look after cases treated with C5 all make the point that the patients are so well after operation; this has been observed by all of us and is I think by far the most important attribute of the drug. It protects the patient against shock. It lessens the impact on him of surgical trauma and this improvement over other cases, even where the blood loss has been accurately or even generously made good, is out of all proportion to the saving of blood. The more severe the operation, the more noticeable this is.

That C5 results in this striking improvement in the level of general condition post-operatively has been noticed by all who have had any appreciable experience of the method. It is this shock-shielding property of the drug which accounts for our using it now in an increasing number of cases where bleeding is not a problem and never has been except as a result of the occasional accident, e.g. gastrectomy and cholecystectomy.

The death-rate certainly has been high, but I believe that, as skill and experience in the use of the method increase and more control is obtained by developments such as Arfonad, the death-rate will fall.

Hypotension produced by C5 or C6 is a method which has many advantages especially for the patient and I believe it has come to stay.

Dr. John Gillies: *Hypotension Induced by Subarachnoid Sympathetic Blockade.*—Hypotension following high spinal anæsthesia was recognized as an aid to surgery thirty years ago by Vehrs, Koster and others. Its value was reaffirmed by Griffiths and Gillies (1948) who also used high spinal block and showed that postural adjustment was the main factor determining the ultimate fall in blood pressure and the primary means of restoring the level in emergency. About the same time Bilsland (1951), working in a neurosurgical unit, was producing an ischæmic operative field by arteriotomy, a method first investigated experimentally by Kohlstaedt and Page (1943) and clinically by Gardner (1946). Later, as a result of the work of Paton and Zaimis (1948) interest was aroused in the possibilities of the methonium compounds. This led to the trial and adoption of pentamethonium and hexamethonium as hypotensive adjuncts in anæsthetic practice by Davison, Scurr, Enderby, Pelmore, Hughes and Wyman. Briefly, the mechanisms involved in the methods mentioned are as follows:

(1) In reduction of blood volume by arteriotomy, blood flow and bleeding are reduced more by virtue of the compensatory vasoconstrictor response than by the fall in blood pressure—a circumstance well known to those who have operated on patients with circulatory depression due to hæmorrhage. This is obviously a state which even when purposely produced and well controlled comes near to being pathological. Circulatory reflex activity usually persists, however, and changes of posture do not, as in the other techniques, produce significant gravitational effects on venous return and blood pressure. Incidentally, in the practice of the method, useful information has been gained regarding the advantages of transfusion by the intra-arterial route.

(2) Total sympathetic paralysis by subarachnoid block or by extradural block as described by Bromage (1951) causes generalized vasodilatation and a fall of blood pressure. By appropriate positioning of the patient pooling of blood in dependent parts, particularly the legs, can be achieved. There is a prolongation of the circulation time, and if the site of operation is raised a little higher than the rest of the body, slow filling and accelerated drainage of the local venules and veins will reduce bleeding from the incised tissues. It should be emphasized that in addition to sparing the patient's blood, total sympathetic paralysis also prevents loss of fluid by the skin—a not inconsiderable factor. By a single intrathecal injection full sensory and sympathetic block along with a safely limited motor paralysis may be simultaneously obtained. When the technique is properly managed the upper intercostal motor nerves and the phrenics are not affected. Thus respiration should remain spontaneous and be an indication of the state of the respiratory centre and the medulla. This important and reliable guide is not always available when muscular relaxation depends on curarization.

(3) In total autonomic blockade all ganglia are involved but in varying degrees. The effects therefore are complex compared with those of spinal and extradural block in which only the sacral parasympathetic segments are affected in addition to the sympathetic outflow. In ganglionic blockade the agent is distributed by the blood stream, a method which is less positive than the direct application of an analgesic drug to the preganglionic sympathetic fibres in the subarachnoid or extradural spaces. This may account in part for the greater certainty of direct nerve block in producing hypotension in and for a predictable period of time.

All the foregoing techniques are practised in our surgical units but for the present discussion only data concerning hypotension induced by subarachnoid injection will be submitted.

The main groups in a series of 802 operations performed under high spinal block and induced hypotension are shown in Table I. There was a high proportion of vascular cases and many of

TABLE I.—OPERATIONS UNDER HIGH SPINAL BLOCK (802 CASES)

Thoraco-lumbar splanchnicectomy	341	Excision of tumours of colon	47
Lumbar sympathectomy	222	Urological operations	63
Partial gastrectomy	51	Miscellaneous	78

these were poor risks who provided a severe test for what in 1946 was generally considered to be a dangerous procedure. The records which follow later may seem poor or, at least, unimpressive but they can bear comparison with those associated with conventional anæsthetic procedures applied to similar cases.

Although planned lowering of blood pressure is not essential in abdominal surgery the principle was tested in this field in order to ascertain its limits and particularly to discover any ill-effects that might develop as a result of prolonged circulatory depression. For operations in this region spinal anæsthesia may still be chosen as much for its undeniable advantages of good muscular relaxation and sensory blockade as for its hypotensive effect but the last-named does make an appreciable difference in a difficult case such as gastrectomy for a posterior ulcer of the stomach which has invaded the pancreas.

The miscellaneous section includes stellate ganglionectomy, radical mastectomy and pelvic exenteration.

Mortality.—Most of the avoidable mortality occurred early in the series mainly as a result of inexperience.

There were two deaths during operation (Table II), from both of which salutary lessons

TABLE II.—MORTALITY RELATED TO INDUCED HYPOTENSION

Deaths on table	2	
Post-operative cerebral vascular accidents	4	(4)
Reactionary hæmorrhage	1	
Total	7	

were learnt respecting the management of the hypotensive state. In the first, the danger of anoxic anoxia due to mechanical restriction of pulmonary ventilation was noted too late. The second patient died after getting a small dose of thiopentone (100 mg.) which abolished spontaneous respiration and so deprived the anæsthetist of his main guide as to the adequacy of the circulation to the medulla. Delay in instituting the simple restorative measure of raising the legs and increasing venous return was a costly and avoidable error of judgment.

Four patients who died of cerebral vascular accidents had histories of pre-operative cerebral vascular episodes which would now be considered contra-indications to any hypotensive technique though such accidents also happen when orthodox anæsthetic methods are employed.

There was one fatality associated with reactionary hæmorrhage.

Morbidity.—Three other cases of reactionary hæmorrhage recovered (Table III).

TABLE III.—MORBIDITY RELATED TO INDUCED HYPOTENSION

	In operations for hypertension	In other operations	Total
Reactionary hæmorrhage	–	3	3
Cerebral vascular accidents	6 (5)	1	7
Coronary thrombosis	1	–	1
Ischæmia of limb	–	7	7
Oliguria	3	–	3
Neurological	1	1	2

In the total of 11 post-operative cerebral vascular accidents included in Tables II and III, 9 (4 fatal and 5 non-fatal) occurred amongst 20 patients with pre-operative records of cerebral vascular episodes while only 2 (both non-fatal but including a retinal thrombosis) happened amongst 782 patients without such records.

In this generally unfavourable series of patients the two bogeys—coronary thrombosis and

renal failure—failed fortunately to materialize. The only instance of coronary thrombosis occurred in a patient with a history of angina, who, ten days earlier, had undergone without incident an extensive transthoracic sympathectomy for the relief of his cardiac pain. After being anæsthetized, the procedure including a spinal block, signs of impending circulatory failure appeared. Before operation could be started cardiac massage became necessary and was successful. As this case was proved shortly afterwards to have been one of coronary thrombosis the treatment by cardiac massage may be unique.

There was one other case of cardiac standstill—again in a hypertensive patient about to undergo splanchnicectomy. In this incident the cardiac arrest was related to an asphyxial episode in which the patient under light anæsthesia (nitrous oxide) obstructed a metal-rubber airway by clenching his teeth on it. He had been given a spinal injection but the respiratory obstruction occurred before any attempt had been made to lower the blood pressure by posturing. Immediate cardiac massage was successful and the operation was performed uneventfully a week or two later under the same hypotensive technique planned for the first occasion, namely high spinal block. There is little doubt that when a patient has not had his tissues saturated by ether or some other noxious general anæsthetic and the heart stops during a period of physiological derangement, it can be started again and there need be no ill-effects if this is done at once and effectively.

There were three instances of oliguria (Table III) in the 802 operations—not an exceptional figure even in non-hypotensive anæsthetic procedures. Expectations have been borne out that a low head of pressure does not matter if there is vasodilatation and the flow of blood to the kidney is not significantly diminished in volume. With non-toxic narcosis and adequate oxygenation the renal epithelium remains healthy and resumes its full function when the arterial pressure begins to rise. During high spinal block with the patient's pressure between 60 and 70 mm.Hg, the continued secretion of urine throughout operation has been observed by means of ureteral catheters.

Seven instances of post-operative ischaemia of limbs occurred in 222 lumbar sympathectomies. If a marked degree of arteriosclerosis is present there may be little or no local vasodilatation following vasoconstrictor paralysis and in consequence blood flow which has been precarious before may now cease because of hypotension and lack of pressure from behind. In one case a limb was lost—a result which was probably inevitable but not expected quite so soon. Such ischæmia, however, may also occur with non-hypotensive anæsthetic techniques and we have a record of a patient in whom this happened twice, once after spinal and again after general anæsthesia.

Finally, the neurological morbidity must be mentioned, for this can be serious for the patient and all concerned. Lesions of the cord in association with spinal block are of relatively frequent occurrence but this depends largely on the skill of the administrator. In expert hands the incidence reaches a low irreducible minimum to which chance is the main contributor.

Conclusion.—Induced hypotension must never be a matter of routine and when applied the following criteria must be satisfied beforehand.

(1) It must be of unquestionable benefit to the patient either by sparing his blood in extensive surgery and so protecting him from the risks associated with replacement of blood or by preventing troublesome bleeding in an operation of precision, thereby contributing to its success.

(2) The patient must be physically suitable particularly in regard to the state of his arteries. Conditions affecting transport of oxygen such as anæmia, hypovolæmia, chronic pulmonary disease, and congestive cardiac failure, are definite contra-indications.

(3) The anæsthetist must be skilled.

(4) There must be close supervision and proper management of the patient in the post-operative period until the circulation has returned to normal efficiency.

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SUMMARY OF DISCUSSION

In answer to an enquiry, **Dr. Gillies** said he did not think hypotensive anæsthesia had been an advantage where he had used it in three operations for portal hypertension. **Dr. C. T. Barry** had noticed ileus to be a feature after its use. **Dr. M. H. Armstrong Davison** had abandoned it because he thought the number of deaths on the table had been increased; **Dr. R. Woolmer** thought similarly and preferred combating shock with blood replacement. **Dr. T. C. Gray** was opposed to its use and thought it ought to be restricted to certain cerebral, plastic, and oto-laryngological operations. **Dr. A. R. Hunter** described its use in a series of 50 neurosurgical cases. He thought that at times, for examples in cases of cerebral angioma and meningioma, it made the operation possible.

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Amœbic Granuloma of the Colon

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AMŒBIC granuloma of the colon is a rare disease. The dysentery ward of which I had charge in South India during the years 1942 to 1944 dealt with 363 cases of amœbic dysentery among British troops and, among these, cases of palpable masses involving the colon were only observed in 13 (3·6%). The disease may manifest itself in three ways, a mass in the region of the cæcum which may mimic an appendix abscess, an ulcer in the rectum which mimics carcinoma and a stenosing mass in the course of the colon which precipitates intestinal obstruction. Histologically two forms occur: one shows an infiltration of the gut wall by inflammatory cells and the other a gelatinous exudate, each being accompanied by the pathogenic amœba. It has been suggested that the reaction is due either to secondary infection of the amœbic ulcers or to some toxin formed by the amœbæ.

Surgical intervention.—Three weeks after arriving in India I assisted at an autopsy at a military hospital. The patient had three weeks previously been found to have a tender mass in the right iliac fossa, fever and leucocytosis. Laparotomy was carried out, the appendix though unaffected was removed and the mass was seen to be the thickened cæcum. Three days after the operation bloody stools were passed and active amœbæ demonstrated. Emetine therapy was begun but on the twelfth day the operation wound broke down and on the twenty-first day the patient died of toxæmia. At the autopsy there was a hole in the right iliac fossa in which the thickened cæcum lay bathed in pus.

During the first week I was in charge of the dysentery ward a death occurred in almost identical circumstances. Fever, leucocytosis and a palpable mass led to a diagnosis of appendix abscess. A thickened cæcum was found at laparotomy and an apparently normal appendix was removed. An acute amœbic diarrhœa occurred two days after the operation and emetine was given but death occurred on the eighth day. At autopsy not only was there a massively thickened cæcum but also an extensive gangrene of the large gut. At five points rupture into the retroperitoneal tissues had occurred and there were abscesses up to the size of a plum.

Similar accidents have been described by Gunn and Howard (1931). In one case a man with chronic amœbiasis of fourteen years' duration had a mass excised from the splenic flexure in the belief that it was a carcinoma. Cysts of *Entamœba histolytica* were found in the stools before operation and its vegetative forms in the excised mass. Death occurred from toxæmia on the ninth day. In a second case a Portuguese who had intermittent diarrhœa with blood for twelve years developed a right-sided mass thought to be carcinoma but which was the thickened cæcum. Death followed a palliative ileostomy and the parasites were found in the cæcal wall. Yeomans (1936) described 2 cases in which colonic masses were excised and amœbæ demonstrated. One died and the other recovered with emetine therapy. Mendelson (1939) describes an operation on a Siamese in which a biopsy of the cæcum showed the parasites but he does not mention the outcome of the case. Lindskog and Walters (1946) encountered a mobile cæcal mass in a young seaman known to have had amœbic dysentery. A right hemicolectomy was followed by recovery but no parasites could be found in the tissue. Nevin (1949) treated a soldier, who had both a cæcal mass and an ulcerated granuloma of the rectum, by emetine and during convalescence was compelled to adopt surgical measures for the relief of obstruction by a band. No interference with the gut was necessary, and the patient recovered uneventfully.

In the 9 cases mentioned death occurred in 5, recovery in 3 and the outcome is not known in one. It is evident that surgical interference is fraught with danger.

Medical treatment.—Of the 13 cases encountered in the Indian dysentery ward 1 was the surgical fatality already mentioned, 10 were cæcal amœbomas treated medically, 1 a case of cæcal amœboma who died, and 1 an amœboma of the rectum. The salient features of the cases treated were as follows:

I. J. H. had a three-day history of diarrhœa with blood. On admission his temperature was 99° F., he had nausea, a foul dry tongue and a tender cæcal mass. *Entamœba histolytica* was demonstrated in the tenth stool examined on the second day. He was symptomless and the mass had disappeared before the twelfth daily injection of emetine had been given.

II. A. F. H. complained of two weeks of abdominal pain and loose fæcal stools. His temperature was normal, he had a dry foul tongue and a tender cæcal mass. He had no nausea and no loss of appetite. He passed a loose stool and then a scybalous mass coated with blood in which the parasites were present. This man was evacuated from the hospital while on emetine therapy and with the mass still present.

III. H. E. had a dysenteric attack five months previously with nausea, vomiting and headache. On admission his temperature was normal, his tongue was clean and there was a tender mass in the right iliac fossa. The fourth stool examined showed parasites. After 12 injections of emetine and five days of Quinoxyl by mouth and as retention enemas the mass could still be felt but disappeared during the next week without further treatment.

IV. W. H. W. was admitted to a surgical ward with a six-day history of diarrhœa with blood and a severe pain in the right iliac fossa. He had a tender cæcal mass. Parasites were found in his stools three days later. He was treated in the dysentery ward with emetine for six days and with carbarsone by mouth for nine days. The mass was still present but disappeared with five days' treatment with Quinoxyl.

V. G. H. had five courses of treatment of different types during the previous two months. Four days after the last he had a recurrence of diarrhœa with right-sided abdominal pain. A tender cæcal mass was found which disappeared before the end of a course of twelve days' emetine.

VI. D. J. A previous attack of amœbic dysentery had been treated with emetine only. When admitted he stated he had been passing stools "almost all blood". For a fortnight he passed fæcal stools without parasites and complained at intervals of upper abdominal pain. Typical ulcers and parasites were found at sigmoidoscopy. A full course of emetine and Quinoxyl was given but ten days later a cæcal mass 3 in. in diameter was found which progressed until it was the size of a fœtal head. Pain was so great that morphine was given in grain doses twice daily and a furious bloody diarrhœa continued in spite of treatment of all types. This continued for a fortnight and the mass began to regress, but two weeks later developed to its former size with a further outburst of the dysentery. He was reduced to an emaciated skeleton with bed sores and massive hæmorrhoids. Intravenous saline and blood were given at intervals. Anti-amœbic drugs had been given almost continuously for five months with no more than transitory improvement and were therefore stopped. He slowly recovered and the cæcal mass disappeared spontaneously in about a month. Two months later he was returned to duty by a medical board.

VII. W. K. was admitted with a fifth attack of amœbic dysentery. His temperature was normal, and he had no nausea. There was a large tender cæcal mass. A test purge of saline on the third day in hospital showed precystic forms of *E. histolytica* and later cysts. A course of carbarsone by mouth for fifteen days left him with a fitful diarrhœa and the mass unaltered, but a test purge showed no parasites and a sigmoidoscopy no lesion. He was invalided out of the country and the mass was still present when he left. In spite of the mass he did not feel ill and was up and about during treatment.

VIII. J. F. was admitted for a third attack of dysentery. He had had fluid stools for three months and blood had been present for five days. His temperature was normal, he was emaciated and there was a large tender cæcal mass. Numerous active parasites were present in the first stool examined. He was treated for twelve days with emetine and for five days with Quinoxyl. The tumour disappeared but he relapsed with further dysenteric attacks and was invalided.

IX. C. M. had one attack a year before. He had diarrhœa with blood for four days before admission, but when admitted his temperature was normal, he had no headache or nausea and little pain. There was a large swelling in the right iliac fossa and gross thickening of the sigmoid. After ten days' emetine and five days' Quinoxyl both swellings had disappeared and the stools were normal.

X. R. J. was desperately ill when admitted after being treated for three weeks for presumed bacillary dysentery. His temperature was 102° F., he was emaciated, had bed-sores and a mass filling the right iliac fossa. His liver was 2 in. below the costal margin and X-ray showed a considerable elevation of the right dome of the diaphragm. The stools showed numerous active parasites on the day of admission and emetine therapy was begun. Attempts to aspirate the liver abscess failed. Death occurred on the tenth day. At the autopsy two large liver abscesses were found to be filled with tough material; the cæcum was greatly thickened and gelatinous. In this case the amœboma was the minor complication.

XI. R. H. had been in hospital almost continuously for a year with attacks of amœbic hepatitis and amœbic dysentery. When admitted he was passing blood on formed stools, had no pain and no feeling of illness. A large irregular swelling in the right iliac fossa was not at all tender. He was treated with emetine and Quinoxyl and the swelling disappeared, but he relapsed with active dysentery and was invalided.

XII. J. L. had had four attacks of dysentery at close intervals. Sigmoidoscopy showed a rectal ulcer 4 in. from the anus, almost encircling the bowel and fully an inch across. The tissue adjacent was thickened and indurated and the ulcer itself was not friable. Scrapings showed many active parasites. Emetine was given and the ulcer disappeared in ten days, but the man was admitted to another hospital shortly afterwards with a relapse of the dysentery.

Large ulcers of the rectum which were clinically indistinguishable from carcinoma and due to *Entamœba histolytica* have been recorded by Donald and Brown (1940), Cameron and Collins (1942), Manson-Bahr (1943), and Nevin (1949). In each case rapid cure resulted from emetine therapy. It is of importance that Manson-Bahr's cases arose ten and twenty years respectively after the active disease.

Diagnosis.—In the case of a stenosing tumour causing obstruction surgical measures are bound to be taken and delay in the attempt to establish a diagnosis of amœboma would not be considered. The nature of the lesion can only be discovered by histological examination of the tissue removed

The ulcerating lesion of the rectum, once it is suspected to be of amœbic origin can be confirmed as such by a biopsy or by taking scrapings of the surface.

A swelling in the region of the cæcum can result from a variety of processes, the most frequent of which are probably suppuration originating from the appendix and malignant disease. The following points may assist in making the distinction:

(1) *The history.*—A patient who has had protracted treatment for amœbic dysentery will probably be aware of the diagnosis which was made. Where there has been a slow and insidious progress of the disease the history may be of no assistance. A British sea captain I saw in Persia who was admitted to hospital as a case of congestive heart failure denied ever having had dysenteric attacks and no evidence of the disease was found in his stools, yet at autopsy the whole of the colon showed large amœbic ulcers sealed off by tenacious exudate.

(2) *Stool examination.*—Red blood cells, degenerate pus cells and Charcot-Leyden crystals are suggestive and the cysts or vegetative forms of the amœba are diagnostic. Sigmoidoscopy may assist but only shows the condition of the lower colon. Lesions in the cæcum yield the parasites very readily after a test purge. Magnesium sulphate in 2 drachm doses in warm water followed by a pint of hot tea was given on an empty stomach to a great number of cases of amœbic dysentery and to several of the cases of amœboma without untoward result.

(3) *Pain and tenderness.*—These were present in varying degree in all but one of the cases.

(4) *Mobility.*—The four largest tumours were immovable. Three of the lesser ones were also immovable. The remaining three could be moved fairly freely.

(5) *White Cell Count*:

	Neutrophil polymorphs	Lymphocytes	Eosinophils	Large mononuclears
D. J. ..	4,508	3,220	637	184
	7,076	3,788	1,098	244
	4,512	4,608	384	96
	7,888	5,032	544	136
A. F. H. ..	5,894	2,376	176	264
J. F... ..	2,640	2,064	96	—
R. H. ..	28,800	3,600	360	—
W. H. W. ..	(Total 18,800)			
G. H. ..	8,240	5,390	1,848	208

It will be seen that there may or may not be an absolute polymorph leucocytosis or an absolute lymphocytosis. An absolute lymphocyte count of over 4,000 is unusual in the adult and should arouse suspicion. The progressive rise in the lymphocyte count in D. J. who was gravely ill for many months is of interest.

(6) Temperature is commonly normal or raised only a degree or so, and other constitutional disturbance is usually slight.

Discussion.—Nevin has pointed out that a great number of Europeans were exposed to infestations by *Entamœba histolytica* during the war years when serving in countries where it is prevalent. It is possible that for years to come the occasional individual who has served abroad may present himself with a cæcal or rectal tumour which, in the absence of an awareness of amœboma by the clinician, may lead to a diagnosis of appendix abscess, carcinoma of the colon or carcinoma of the rectum.

Surgical treatment frequently results in disaster whereas medical treatment, though often slow and disappointing, may be expected to lead to a recovery. Awareness of the possibility is essential, otherwise the investigations which will lead to a correct diagnosis will not be initiated.

Diagnosis in areas where the disease is rife is admittedly easy and in India the disease was severe among the incoming Europeans, the vegetative forms of the parasite were plentiful and their discovery a matter of hours or days. In a temperate climate the disease is rare and the parasites likely to be scanty and in the less obvious cystic form. Nevertheless once suspicion is aroused and surgical measures withheld a patient search should show their presence.

[This extract from a thesis submitted for the degree of Doctor of Medicine has been published with the permission of the University of Leeds.]

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Megacolon

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MEGACOLON may be classified into three types:

(1) Obstructive or Symptomatic (Table I).

TABLE I.—TYPE I. OBSTRUCTIVE OR SYMPTOMATIC

Can occur at any age.

Cause: Obstruction to the colon from:

(1) Lesions outside the bowel wall—pelvic tumours.

(2) Lesions in the bowel wall itself or within the bowel lumen.

(*a*) Strictures { Congenital. Inflammatory—diverticulitis. New growth—annular carcinoma.

(*b*) Stenosis { Congenital. Inflammatory.

(*c*) Tumours { Polypoid. Chronic intussusception.

(3) Lesions of C.N.S.—Spina bifida.

Result: Chronic obstruction of large bowel with hypertrophy and dilatation proximal.

Treatment: Relief of obstruction and removal of the obstructing agent.

(2) Aganglionic or Hirschsprung's Disease (Table II).

TABLE II.—TYPE II. AGANGLIONIC OR HIRSCHSPRUNG'S DISEASE

A rare disease of childhood, ultimately fatal if not treated.

Cause: Congenital absence of Auerbach's and Meissner's plexuses in a variable length of bowel.
Lack of co-ordinated peristaltic movement in the affected segment.
Secondary dilatation and hypertrophy of colon proximal.

Sites: Generally lower reaches of sigmoid and upper rectum. May affect longer segments or even the whole colon.

Diagnosis: Barium enema demonstrating narrowed segment.

Treatment: Removal of diseased segment with reconstitution of bowel in continuity.

(3) Simple (Table III).

TABLE III.—TYPE III. SIMPLE

(Also known as idiopathic megacolon, pseudo-megacolon, dolichocolon, colon inertia)

Can manifest itself at any age.

Commonly misdiagnosed as Hirschsprung's disease in childhood.

Cause: Congenital elongation of the sigmoid colon with its mesenteries followed in time by dilatation and hypertrophy due to slowly acting, benign and inconstant obstructing factors:

(1) Faulty bowel habits: Chronic constipation.

(2) Bulky cellulose-containing diets (Eastern Europe).

(3) Repeated attacks of subacute and chronic volvulus.

(4) Atony of bowel in old age.

Treatment:

(1) *Medical*—Correction by enemas, drugs, proper training in bowel habits.

(2) *Surgical*—Resection of dilated segment.

I wish to discuss only the simple type of megacolon, the fundamental starting point of which is the congenitally elongated sigmoid colon found in 20 to 25% of individuals.

Embryology of the left half of the colon: The hind-gut in fœtal life has a long mesentery similar to that found in all species of monkeys and anthropoid apes, which partially disappears at birth, the descending colon becoming fixed to the left side of the posterior abdominal wall. In the first four months of extra-uterine life, a striking feature is the short length of the descending colon as compared with the sigmoid.

The sigmoid colon at birth measures 10 in. to 1 ft. appearing in contrast to the rest of the infant colon as an enormous loop, the remainder of the colon being very short in comparison. The colon gradually lengthens at the expense of the sigmoid and by the fourth month of extra-uterine life, the sigmoid loop assumes the adult pattern measuring 6 in. on an average, the remaining original 4 in. being "taken up" by the descending colon. From the standpoint of its development and comparative anatomy, the sigmoid will vary greatly in its length and fixation. Heller many years ago called the long congenital type of sigmoid "Megasigmoideum congenitum".

The causes of the obstruction (Table III) usually operate inconstantly for a long period, even many years, and the megacolon may not manifest itself symptomatically till old age.

A simple megacolon conforms to one of two distinct patterns (Bodian):

(*a*) *The tubular type* (Fig. 1).—A long redundant sigmoid which is dilated and hypertrophied as far as the anal sphincter. This long loop of bowel stretches up towards the right upper abdominal quadrant and the dilatation extends proximally to involve more or less of the remaining large bowel.

(*b*) *Terminal reservoir type* (Fig. 2).—An enormous dilatation of the sigmoid and rectum stopping below at the anal sphincter and above at the junction of the sigmoid and descending colons, where an acute kink may be seen. Beyond this point, the calibre of the colon is generally normal.

FIG. 1.—Barium enema showing "tubular" type of simple megacolon in male child aged 4 years.

FIG. 2.—Barium enema showing "terminal reservoir" type of simple megacolon in male aged 46 years.

Simple megacolon in childhood: Table IV gives the presenting symptoms in order of frequency in childhood.

TABLE IV.—SIMPLE MEGACOLON

Presenting Symptoms in Childhood in Order of Frequency

Age from birth onwards.
Sex ratio equal.

(1) Fæcal impaction presenting symptomatically as:
  (*a*) Diarrhœa.
  (*b*) Obstruction.
(2) Abdominal distension, "Pot belly".
(3) Encopresis—soiling of clothes.
(4) As an acute emergency:
  (*a*) Acute retention of urine.
  (*b*) Acute volvulus.
  (*c*) Anal fissure.
(5) Chronic volvulus.

TABLE VI.—SIMPLE MEGACOLON

Presenting Symptoms in Order of Frequency in Adults

(1) Acute volvulus—emergency.
(2) Repeated attacks of chronic volvulus—a clinical entity.
(3) As a colitis.
(4) As a malignant disease of the colon.
(5) Intractable constipation (rare).
(6) Fæcal impaction.
(7) Abdominal neurosis.

Examination will show a miserable child with distended abdomen, pasty complexion, irritable. Visible peristalsis may be seen, and the loaded colon palpated. The rectum is blocked with fæcal matter, often with soiling around and sometimes an anal fissure is present. Diarrhœa is a common complaint and sometimes obstructive symptoms are present, both being due to fæcal impaction.

A barium enema is diagnostic, but should be avoided unless the diagnosis is in doubt or Hirschsprung's disease is suspected. If a barium enema is given, only a thin emulsion should be used or, in babies, lipiodol, as there is a grave risk of producing severe barium impaction.

The differential diagnosis from Hirschsprung's disease is set out in Table V, modified after Bodian *et al.* (1949).

TABLE V

	"Hirschsprung"	Simple
Onset	Neonatal	Later
Sex	Boys predominate	Many girls
Motions	Pellets, ribbons	Large diameter. Hard
Encopresis	No	Common
Abdomen	Distension ++ Visible peristalsis, gross, audible. Distended colon palpable, gas cushion	Distension less. Visible peristalsis. Less marked if present. Distended colon, palpable, firm
P.R...	Anus clean. Calibre and sphincter normal. Rectum empty. Odd pellets	Anus dirty. Possible cracks or stenosis. Fissures
Retarded growth ..	++	Rare ?
Radiography ..	Narrow lower segment	Terminal reservoir or tubular dilatation
Complications ..	Acute obstruction Perforation of stercoral ulcers	Retention of urine Volvulus—acute or chronic
Pathology	Agenesis of ganglia in distal segment	Distal segment normal
Treatment	Operative	Non-operative ?

The presenting symptoms of megacolon in an adult fall into seven groups (Table VI).

Group I.—Acute volvulus is familiar to you all. Carnes Weekes (1931) collected 68 cases of acute volvulus of a megasigmoid—15 of which had had a total of 23 previous operations for relief with a 24·5% mortality.

Group II.—Chronic volvulus I believe to be a clinical entity with a distinct symptomatology. The chief complaint is of attacks of severe spasmodic pain with varying and often gross distension of the abdomen due to the enormous gas-filled bowel. The pain and distension come on at any time, the patient obtaining relief by the adoption of different bodily attitudes, as the knee-chest position, or lying face downwards. The distension is always marked during the attacks and complaint is made of inability to fasten clothes round the waist. In some the distension tends to become a chronic feature with little or no pain and the patient will carry on with normal duties. Constipation is rare, but the passage of flatus gives relief. Many of these patients are seen in a latent period, and a carefully taken history is important. Volvulus is a dynamic and not a static condition. Many of these unfortunate people receive scant sympathy and are frequently diagnosed as suffering from abdominal neurosis, finally ending up in the hands of a psychiatrist. The symptomatology is exact, however, and chronic volvulus a definite syndrome.

The following case is illustrative: A young healthy single woman of 23 years consulted me with all the symptoms. She was sent up with a diagnosis of anxiety neurosis but a careful history and barium enema gave the diagnosis, showing a megacolon of the tubular type (Fig. 3) and she readily agreed to operation for relief. Fig. 4 is a photograph of the sigmoid taken at operation prior to resection. Fig. 5 a barium enema four months after operation. She obtained complete relief.

Group III.—Other cases suggest some form of colitis, and are often diagnosed and treated as mild cases of ulcerative colitis, prior to a barium enema and sigmoidoscopy. The chief complaint is of pain relieved by the passage of flatus and watery blood-stained stools, the blood coming from the traumatized sigmoid mucosa from fæcal impaction and venous stasis accompanying attacks of distension. Clinical examination will reveal little beyond a palpable tender mass in the left iliac fossa. The diagnosis is made by barium enema. The X-ray of the terminal reservoir type (*see* Fig. 2) was of a medical man aged 46 who consulted me with the above symptoms.

Group IV comprises those who simulate carcinoma of the colon. In this group the patient is usually in the fifth or sixth decade, with the general appearances of malignant disease and lassitude and loss of weight. Alternating constipation and diarrhœa, vomiting, loss of appetite and abdominal pain confined to the flanks are the chief complaints. Gross fæcal impaction is nearly always present and is the cause of the symptoms, although attacks of chronic volvulus also occur.

I have treated 3 such cases all over the age of 60, the presenting symptoms all suggesting carcinoma of the colon.

A woman of 65 was admitted to hospital as an emergency with distended abdomen and a diagnosis of acute or chronic obstruction due to carcinoma of the colon. She gave a three months' history of loss of weight, lassitude, alternating constipation and diarrhœa with blood-stained stools. The cause was fæcal impaction in a simple megacolon, proven by barium enema (Fig. 6). She was treated by colonic irrigations and rehabilitation of bowel habit.

Intractable constipation (Group V) is rarely the sole complaint in adults; they more often complain of diarrhœa due to the irritation associated with fæcal impaction (Group VI) and I have mentioned that a diagnosis of abdominal neurosis (Group VII) is often made in those who suffer from attacks of chronic volvulus until investigation shows the true state of affairs.

FIG. 3.

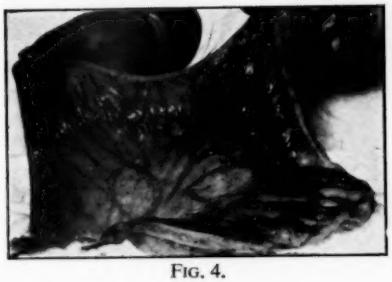

FIG. 4.

FIG. 3.—Barium enema showing simple megacolon of "tubular" type in female aged 23 years.

FIG. 4.—Photograph of sigmoid colon at operation prior to resection (for X-ray *see* Fig. 3).

FIG. 5.—Barium enema four months after resection of sigmoid colon shown in Figs. 3 and 4.

FIG. 6.—Barium enema showing simple megacolon in a woman of 65 presenting with fæcal impaction.

FIG. 5.

FIG. 6.

Treatment may be medical or surgical.

Medical treatment should be tried in cases of simple megacolon in children except for an emergency such as acute volvulus.

I have the child admitted to hospital and the loaded bowel cleared with *repeated* colonic lavage. Disimpaction of fæces under anæsthesia may be necessary prior to the lavage. Any anal fissure should be treated by the application of 2% nupercaine ointment to the anus, prior to an evacuation of the bowels. After a few days have been spent in clearing the bowels, *training* to restore a normal bowel habit should be instituted. This is the most important part of the treatment. The child must be taught to go to stool regularly and at set times. In the early period of training, the bowels should be assisted by mild aperients as liquid paraffin or milk of magnesia. I have had no experience in the use of Prostigmin or spinal anæsthesia.

FIG. 7.

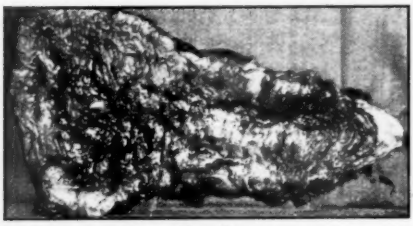

FIG. 8.

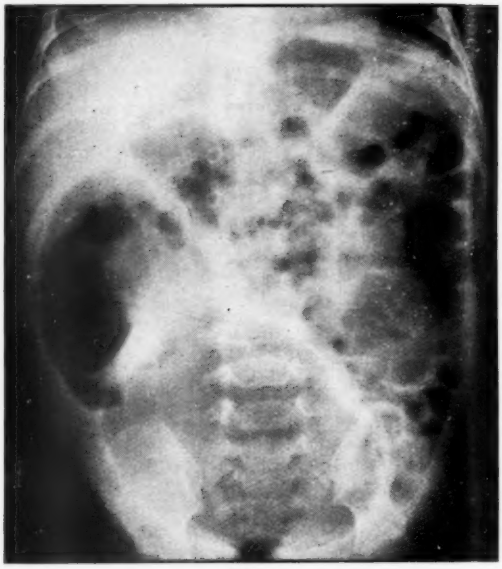

FIG. 9.

FIG. 7.—Barium enema showing simple megacolon in female child aged 2½ years.

FIG. 8.—Photograph of resected specimen of colon (X-ray Fig. 7). Note traumatized mucosa which gave rise to severe hæmorrhage.

FIG. 9.—Straight X-ray of abdomen in infant of 6 months showing acute volvulus of sigmoid colon.

These measures should continue for three months—after this time the child becomes bowel conscious and repeated enemata may cause psychological trauma. If after three months there is no real response, then a resection of the redundant sigmoid should be considered. I agree with Lee *et al.* (1950) that speedy cure can be obtained by resection in "selected" cases and, by removing the redundant bowel, a larger area of water-absorbing mucosa is removed, resulting in a softer, easily evacuated stool.

The cases of simple megacolon I have treated in children are listed in Table VII.

TABLE VII.—SIMPLE MEGACOLON IN CHILDREN—*Aylesbury*: 1940–1950

Presenting symptoms		Age in years	Sex	Treatment
Acute volvulus	2	½	M	Relief by rectal catheter
		2¾	F	Operation. Untwisting
Severe rectal hæmorrhage. Child *in extremis* on admission	1	2½	F	Resection after medical treatment
Chronic volvulus	1	3	M	Resection after several attacks
Fæcal impaction	6	8	M	Medical
		8	F	Medical
		4*	M	Resection†
		8	M	Medical
		6*	M	Resection†
		4	M	Medical

*Admitted as emergencies with acute retention of urine.
†After failure of medical treatment.
Total cases 10—all were cured.

I have resorted to resection on four occasions, twice after failure of medical treatment, once for chronic volvulus, and once for severe rectal hæmorrhage. This last case, a child aged 2½ years, was

admitted *in extremis* from blood loss as a medical emergency and referred to me by my colleague Dr. D. MacCarthy for operation. Fig. 7 is a barium enema prior to resection and Fig. 8 a photograph of the specimen measuring 8 in. showing the traumatized mucosa.

In acute volvulus in infants, it may be possible to effect relief by the insertion of a soft rubber catheter passed via a proctoscope thus avoiding operation—provided that gangrene of the twisted loop can be excluded. The catheter should only be passed by a surgeon in theatre as perforation of the bowel can easily occur. Fig. 9 is a straight X-ray of the abdomen in an infant of six months with an acute volvulus of the sigmoid.

In adults: *Medical measures* similar to those adopted for the child should be tried with a non-irritant low residue diet and are indicated in: (1) All cases of fæcal impaction. (2) Those cases presenting as a colitis. (3) Old age. Provided always that attacks of acute or chronic volvulus have not occurred and that the possibility of malignancy is entirely excluded.

Surgical removal of the redundant bowel performed in one stage with resection and end-to-end anastomosis should always be done in: (1) *Acute volvulus*. Untwisting alone is insufficient, and attempts to anchor the redundant bowel useless and dangerous. The only safeguard against a further attack is adequate resection. (2) *Subacute and chronic volvulus*. Here resection will cure the patient and remove the ever-present fear of an acute volvulus.

My series of cases of simple megacolon treated in Aylesbury in the past eleven years are set out in Table VIII. The mortality rate of acute volvulus was 50%.

TABLE VIII

Cases of Simple Megacolon in Adults *Aylesbury*, 1940–1951		Acute Volvulus, 17 *Aylesbury*, 1935–1950	
(1) *Medically treated*:		*Site* : Sigmoid .. 14	
Fæcal impaction	3	Cæcum .. 3	
Simulating colitis	2	*Of these*: 3 were moribund on admission, dying shortly afterwards.	
(2) *Surgically treated*: Paul Mickulicz ..	3	1 (infant) relieved by flatus tube.	
One-stage resection ..	7	13 emergency operation for relief.	
Subacute and chronic volvulus ..	7	6 died as a result of operation.	
Simulating carcinoma of the colon ..	3	Mortality rate 50%.	
Total 15 Mortality nil.			

SUMMARY

The ætiology of simple megacolon is that a congenital long sigmoid of the fœtal type is subjected to long, slowly acting and inconstant obstructing factors, punctuated by periods of automatic relief. Symptoms may arise at any age, with complications which may prove lethal unless promptly treated.

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Congenital Rectal Stenosis

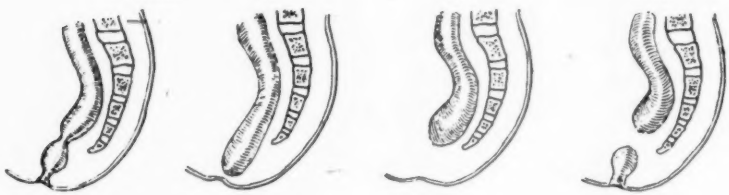
By H. W. A. BARON, M.Chir., F.R.C.S.

THE orthodox view of the development of the rectum is that it arises from the posterior part of the primitive cloaca, as the result of the downgrowth into this of a septum. The cloacal septum finally meets the integument in the region of the future perineum, and the smaller posterior part of the cloaca thus separated gives rise to the rectum, the anterior part to the terminal portions of the urinary and genital ducts. Keith (1932), Wood-Jones (1904), and Denis Browne (1951) find this description inadequate to explain the developmental anomalies in the region, and hold that all the cloaca below the mesonephric ducts is destined for the genito-urinary system, while the rectum in this part arises as a progressive caudal extension of the hind-gut. The rectal tube in the 42 mm. embryo presents two swellings in its lower part, each with a definite constriction at its upper end, the bulbus terminalis and the bulbus analis (Ladd and Gross, 1941). The upper swelling, the bulbus analis, corresponds in position to the rectal ampulla of the adult.

Ladd and Gross (1941) describe four types of congenital ano-rectal stenosis or atresia, which are shown in Fig. 1. This is taken from their book together with the incidence of the different types (Table I). 55% of these cases are associated with fistulæ.

TABLE I.—DISTRIBUTION OF ANAL AND RECTAL ABNORMALITIES, IN MALES AND FEMALES

Type	Males	Females	Total
Type 1	13	11	24
Type 2	7	2	9
Type 3	75	87	162
Type 4	12	7	19
Totals	107	107	214



Type 1 Type 2 Type 3 Type 4

FIG. 1.—Types of anal and rectal abnormalities. Type 1, Stenosis at anus or at a point several centimetres above the anus. Type 2, Imperforate anus. Obstruction due to persistent membrane. Type 3, Imperforate anus. Rectal pouch ending blindly some distance above anus. Type 4, Anus and anal pouch normal. Rectal pouch ends blindly in hollow of sacrum.

Table I and Fig. 1 from Ladd, W. E., and Gross, R. E. (1941) "Abdominal Surgery in Infancy and Childhood". By kind permission of the publishers, Messrs. W. B. Saunders Company, Philadelphia.

Type I.—Stenosis occurring at, or 1–4 cm. above, the anus. The higher stenoses are probably due to incomplete development of the upper part of the bulbus analis.

Type II.—Membranous imperforate anus due to persistence of the anal membrane. This represents an arrest at the eighth week of development.

Type III.—The anus is imperforate, and the rectum ends blindly 3–4 cm. above it. There may or may not be external sphincter musculature present round the skin dimple. Keith (1932), Wood-Jones (1904), and Browne (1951) criticize the orthodox theory of development on the ground that it does not explain this type of anomaly, but a possible explanation is that the normal process of resorption of the tail-gut is extended to involve the lower portion of the hind-gut. This has been shown to occur in ostrich embryos (Boyden, 1922).

Type IV.—A normal anus and anal pouch, which includes the rectum of the adult to the ampulla, the remaining rectum ending blindly 3–4 cm. above the anus. There may be a fibrous cord connecting the two pouches.

Wood-Jones' theory of progressive caudal extension fails to explain this discontinuity, which appears to be due to concrescence at the constriction at the upper limit of the bulbus analis.

Stenosis of the colon is a very rare form of congenital intestinal stenosis. It is thought to be due to a failure of the bowel to recanalize adequately after passing through the normal solid stage in development. The lumen in this anomaly varies much in size, and histologically there may be marked heaping-up of mucosa and submucosa, or the picture may vary little from the normal except for size.

Case Report: A boy of 17, seen May 1951, at St. Margaret's Hospital, Epping.

History.—When he was 36 hours old he was admitted to a large London teaching hospital, having passed no stool since birth. He was distended and vomiting. Examination revealed a normal anus. 1 in. from the anus there was a very tight stricture. At laparotomy the sigmoid colon was found to be represented by a fibrous cord. A colostomy was established in the lowest part of the colon which had a normal lumen.

He was seen again at 5 years of age, when it was found that the stricture was now 7·5 cm. from the anus, but would not admit any instrument. The lower end of the colostomy had retracted in, so it was not possible to get barium down it. He did not report again, as requested.

On examination (May 1951)—A well-nourished youth. Left inguinal colostomy functioning well. Left testicle not in scrotum. The anus was normal, and digital examination revealed normal tone and good voluntary contraction of external sphincter and levator ani muscles. A full-sized sigmoidoscope could be passed for 9 cm. when the lumen narrowed abruptly. Rectum of normal calibre, mucosa normal.

A barium enema confirmed the presence of a normal rectum. A close examination of the film (Fig. 2) shows that it tapers gradually to a very small size, and then there is a large sac filled with mucus thinly coated with barium; above this again is the defunctioned sigmoid loop. The sac is in fact proximal to a diaphragm type of stricture of the upper rectum.

Operation (June, 1951).—The colostomy was excised, and a cord-like sigmoid found running down below the peritoneal reflection. Division of the peritoneum and traction on the sigmoid brought into view a large dilated sac which was thought to be the upper end of the rectum. When it was opened, however, it was found to communicate with the rectum only by a very tiny hole in a diaphragm type of stricture (Fig. 3). Section of the rectum was carried out well below the stenosis, and the sigmoid loop excised as its tiny size

was probably due, in view of the findings as a baby, to congenital causes rather than defunctioning. After thorough mobilization of the descending colon and splenic flexure, an anastomosis to the rectal stump was achieved without tension. The left testicle was found to be retained in the abdomen and was removed. A catheter cæcostomy completed the operation.

The first week of convalescence was marked by some fever and a discharge from the drain to the anastomosis. This cleared up after ten days, and he went out after a month with all wounds healed.

He was last seen twenty months after the operation, when he had perfect bowel control and one normal stool a day. There was hardly any trace of the anastomosis on sigmoidoscopy.

Sections of the sigmoid show that it possesses all coats and has normally developed nervous plexuses. There were two or three blind reduplications of this part of the bowel. A section across the diaphragm stricture shows this to be formed by mucosa and submucosa only.

This case is a little difficult to classify. From the history of the findings at his operation as a baby, it seems that the condition of the sigmoid was, in part, congenital. The rectal condition represents a severe Type I stenosis, presenting as, and to be considered for purposes of treatment as, Type IV.

FIG. 2.—Barium enema on the reported case. Note the normal rectum, the thready sigmoid below the colostomy and the large sac proximal to rectal stricture.

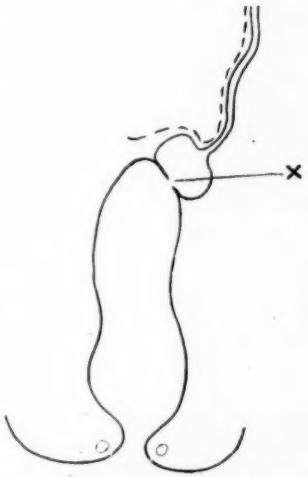


FIG. 3.—Diagrammatic representation of the state of affairs found at operation. The dotted line represents the peritoneum. Cord-like sigmoid runs down from the colostomy to end below the reflection in a large sac. This communicates with the rectum through a tiny hole in a diaphragm-type of stenosis (X).

Clinical presentation.—Those cases in which there is a vent, however small, for gas and meconium may live for long periods, or even indefinitely (Browne, 1951). When there is complete atresia without a fistula, the babies are seen in the first day or two of life, acutely obstructed. In these cases Wangensteen and Rice (1930) advise that a plain X-ray should be taken with the child held upside down for five minutes, when intestinal gas will outline the limits of the blind end. This method is only sure of success after twenty-four hours of life, as before then gas may not have reached the lower reaches of the bowel.

Treatment.—In Types I and II stenoses of moderate severity dilatation with or without incision is usually all that is required.

Types III and IV cases pose the problem both of primary and definitive treatment. Wangensteen is quoted as saying that a perineal approach to the blind sac should always be made because the mortality rate of this is 25%, and that of colostomy 50%. The figures are accurate but the conclusion misleading because colostomy was reserved for cases too ill to stand a perineal dissection, or for cases in which it had failed. The blind end is too high to be reached from the perineum in a quarter of Type III and a third of Type IV cases (Ladd and Gross, 1941), and these authors now recommend colostomy as the primary treatment in Type IV cases. Denis Browne regards "the traditional dissection upwards through the pelvis as a disastrous mistake which should never be made" and recommends colostomy in the first instance, of the spurred variety and in the middle of the transverse colon. The distal loop requires a good deal of attention if fæcal impaction or prolapse are subsequently to be avoided.

Definitive treatment in the Type III cases is limited in its results by the frequent absence of an effective sphincter musculature, especially when what there is has been further embarrassed by perineal dissection. Mayo and Rice (1950) state that only 50% of all their patients could be classified as having a good result, and these "successes" included a good number of patients with permanent

colostomies. Type IV cases have usually been treated by perineal dissection also, and with similar results but a higher primary mortality. The case of the boy reported here emphasizes the feasibility in this type of a formal anastomosis by the abdominal approach, as in restorative resection for carcinoma. It is clear that the distal rectum develops very satisfactorily by itself as it were, and becomes a very useful structure in time. Only one report of an abdominal anastomosis has been traced in the literature, that of Patton (1949). He had a Type IV case in which he did a colostomy on the second day of life, and at fourteen months he carried out an abdominal anastomosis with success. This seems a little early, but the operation appears to be perfectly feasible by the age of 6 or 7 years. The disadvantage is the effect on the child and parents of a colostomy for those years, but if it could be closed before the child went to school it would not be insupportable. Certainly the results of anterior anastomosis, restoring normal rectal function, are infinitely better than those too frequently seen after perineal dissections—"an unsatisfactory colostomy in an uncontrollable place, with bouts of obstruction due to contraction of scar tissue around the opening alternating with bouts of incontinence when this opening is dilated" (Browne, 1951).

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Radical Excisions for Cancer of the Transverse Colon and of the Left Side of the Colon

By STANLEY O. AYLETT, M.B.E., F.R.C.S.

IN 1948 Gilchrist and David published the follow-up results of a series of cases operated upon for carcinoma of the cæcum and of the left side of the colon. Although the series is admittedly small the results are worthy of careful note. Of the cases suffering from carcinoma of the cæcum, in which the lymph glands were found to be involved on pathological examination of the excised specimens, just over 70% were alive and well at the end of five years and were clinically free of the disease. In the comparable cases suffering from cancer of the left side of the colon only 32% were similarly free of disease.

Now there is no reason to suppose that carcinoma of the left side of the colon is less amenable to surgical extirpation than carcinoma of the right side and the poor results in respect of the left side in this series must be attributed to the limited excisions carried out as the current surgical practice at the period during which they were treated.

The wide field of lymphatic and lymph gland involvement in cancer of the colon is well known. In carcinoma of the right colon, where hemicolectomy is carried out as a routine treatment, this extensive field is extirpated, but in cancer of the left side of the colon comparably more limited excisions are still routine surgical practice although of recent years their extent has increased.

In theory the only operation that fulfils the demands of radical extirpation of the disease, where cancer exists in the left colon, is a left hemicolectomy such as is advocated by Grinnell and Hiatt (1952), and I suggest that this resection be adopted by surgeons versed in the intricacies of colonic surgery, at least for patients in the younger age group.

Throughout the cæcum and the colon the distribution of the lymph glands and of the lymphatics follows a similar pattern. Glands, connected by a network of lymphatics, are situated along the mesenteric margin of the bowel, along the smaller arterial branches coursing to any particular part of the colon, and around the trunk and origin of the main arteries, each of which contributes the supply of blood to a very large region of the colon.

In cancer the glands closest to the growth are not necessarily those that first become infiltrated with carcinoma, and with a small and apparently early neoplasm it is not unusual to find distant glands involved. The glands between these and the primary carcinoma have, in some way, been by-passed. It is likely, therefore, that in many cases the glands around the origins of the vascular trunks will be involved and we must plan our surgery so that these are removed. It is not possible to dissect the glands away by stripping them off the vascular pedicle which they surround, nor is it good surgical practice when dealing with cancer. The artery must be divided at its origin with the tissues that surround it.

The division of the artery so far away from the colon deprives an extensive region of the large bowel of its blood supply and it is this and its corresponding mesocolon that have to be removed in radical operations for the cure of cancer.

In dealing with a growth in the cæcum the surgeon raises the whole of the right side of the colon and the terminal ileum, and the ileo-colic and right colic arteries, prior to excising the colon and carrying out an anastomosis, are ligated at their origins. A very radical excision is thus completed (Fig. 1).

The comparable operation on the left side of the colon consists in raising the latter, mobilizing the splenic flexure and upper part of the rectum and ligating the inferior mesenteric artery at its origin from the aorta. By this ligation the blood supply of these parts of the bowel is cut off and they have, therefore, to be excised. Continuity is established by anastomosing the distal portion of the transverse colon to the middle third of the rectum, the former deriving its blood supply from the left branch of the middle colic artery and the latter from the middle hæmorrhoidal arteries.

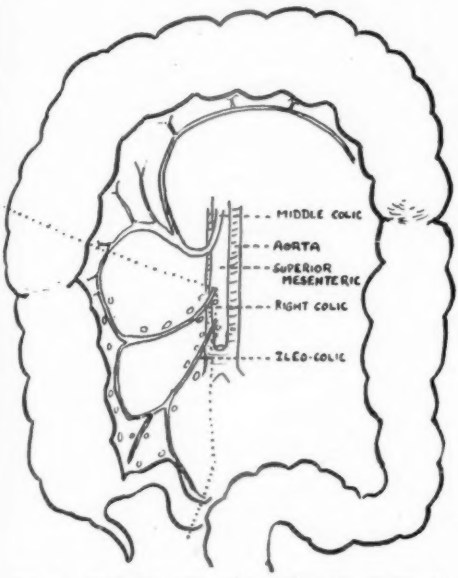

FIG. 1.—Essential area of resection in cancer of cæcum and ascending colon.

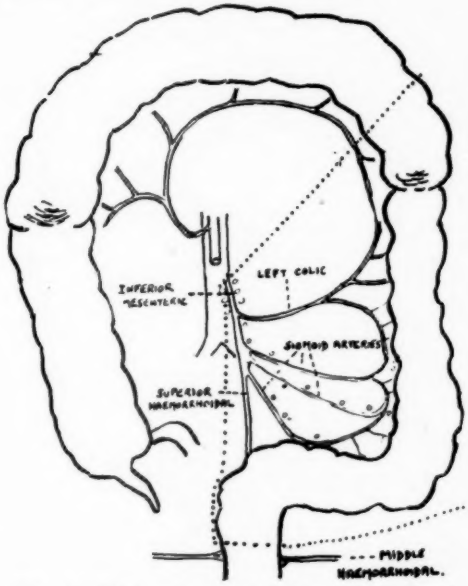

FIG. 2.—Ideal area of resection in cancer of descending and pelvic colon.

In most cases this extensive operation presents few practical difficulties. It cannot be carried out in the few cases where the transverse mesocolon is congenitally deficient. Neither is the operation desirable in the old or enfeebled, but many cases of cancer of the colon are seen in a relatively young age group; it is in these that the surgeon should aim at the widest possible extirpation of the zone of tissue potentially infiltrated with the disease.

Left hemicolectomy is especially essential in the treatment of the growth which involves the adjacent regions of the descending and pelvic colons (Figs. 2 and 3). Here the lymphatic spread may be upwards to involve the glands around the branches of the left colic artery or downwards to infiltrate those about the sigmoidal vessels, so that no lesser operation is likely to cure the condition once lymphatic spread has commenced.

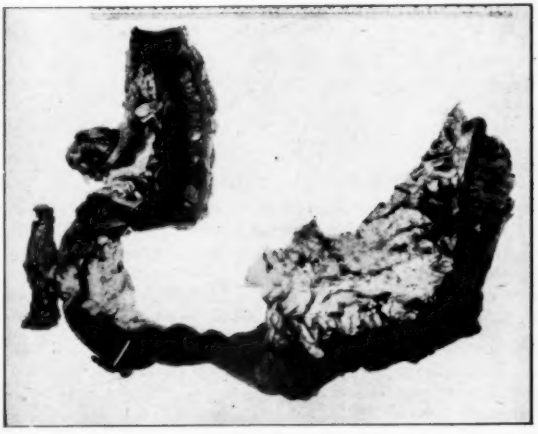

FIG. 3.—Specimen showing the distal portion of the transverse colon, the pelvic and sigmoid colons and the upper part of the rectum removed in the operation of left hemicolectomy.

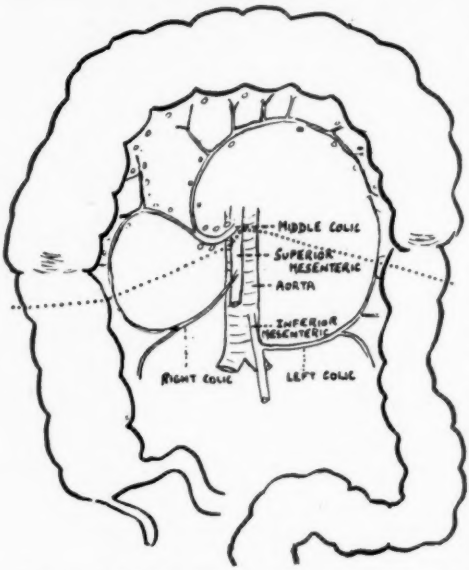

FIG. 4.—Essential area of resection in cancer of transverse colon.

In cancer of the transverse colon the same principles apply (Fig. 4). The glands around the origin of the middle colic artery may well be involved in any growth affecting this region and it and the tissues about it must be excised. Ligation of the artery at the point of its origin from the superior mesenteric artery deprives the whole of the transverse colon of its blood supply and it and its mesocolon have, therefore, to be removed. Continuity is established by anastomosing the upper ends of the ascending and descending portions of the colon. No lesser excision is compatible with the principles of radical cancer surgery.

The radical extirpation of growths involving the splenic flexure presents a difficult problem. Its blood supply is derived from two sources, the left branch of the middle colic artery and the ascending division of the left colic artery. It is possible, therefore, that in cancer of this region the glands around the origin of the middle colic and inferior mesenteric arteries, of which latter the left colic artery is a branch, may be involved in the spread of the disease. Ligation of these vessels would interrupt the supply of blood to the upper part of the rectum, the whole of the left side of the colon and the transverse colon, so that these regions would have to be excised, continuity being established by anastomosis of the upper end of the ascending colon to the middle third of the rectum. The excision, although practicable, is an extensive one, and would be bound to leave the patient with excessive frequency of bowel action as a result of the removal of such a large part of the water-absorbing region of the colon. I have, therefore, not practised this excision, but in cases of carcinoma of the splenic flexure I ligate the middle colic artery at its origin and the left colic where it arises from the inferior mesenteric artery. The transverse colon, the splenic flexure and the descending colon with their mesocolons are then excised, and the upper end of the ascending colon is anastomosed to the cut end of the rectum to restore continuity. This is a far wider excision than is usually carried out in dealing with growths in this region (Fig. 4).

The operations described have been carried out as one-stage procedures without a preliminary colostomy or cæcostomy. Post-operative progress has been similar to that following less wide excisions, and the ultimate frequency of bowel action is but slightly increased even following a left hemicolectomy.

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[*March* 11, 1953]

MEETING AT ST. MARK'S HOSPITAL, CITY ROAD, E.C.1

The following Cases and Specimens were shown:

Ulcerative Colitis Showing Three Strictures (Subtotal Colectomy Specimen).—Mr. W. B. GABRIEL.

(1) Ulcerative Colitis Associated with 4 Malignant Tumours (Subtotal Colectomy Specimen). (2) Ulcerative Colitis Associated with Pseudopolyposis (Right Hemicolectomy). (3) Ulcerative Colitis (Total Colectomy).—Mr. C. NAUNTON MORGAN.

Two Cases of Ileostomy Dysfunction.—Mr. H. E. LOCKHART-MUMMERY.

Ileostomy Appliances.—Mr. BRIAN COUNSELL.

Restorative Resection and Partial Cystectomy for Carcinoma of the Colon (5 Cases).—Mr. H. R. THOMPSON.

Rectal Carcinoma Following Subtotal Colectomy and Ileo-rectal Anastomosis in Familial Polyposis.—Mr. J. C. GOLIGHER.

Demonstrations: An X-ray demonstration of various forms of carcinomata (approximately some 50 types) was given by **Dr. Norman P. Henderson,** Senior Radiologist, St. Mark's Hospital.

Dr. Cuthbert Dukes and **Mr. H. J. R. Bussey** gave a demonstration of specimens in the Pathological Department.

JOINT MEETING No. 3

Section of Neurology with Section of Orthopædics and Section of Pædiatrics

Chairman—MACDONALD CRITCHLEY, M.D., F.R.C.P.
(President of the Section of Neurology)

[*February* 5, 1953]

DISCUSSION ON INFANTILE CEREBRAL PALSIES

Dr. R. M. Norman (Stoke Park Colony):

Pathology and Ætiology

During the past sixteen years I have examined post-mortem 45 brains of individuals who had suffered from cerebral palsy and I hope to further the purposes of this discussion by analysing briefly the results of this work and by describing some of the commoner pathological findings. This material is derived mainly from certified mental defectives at Stoke Park Colony, only 5 cases having come from general hospitals. As regards the method of clinical selection, all the patients in this group showed severe disorders of motor function and I have not included any examples of the mild generalized spasticity so frequently found in low-grade mental defectives and for which, so far as I am aware, there is no recognized pathological basis. As will be seen in Table I, the younger age groups predominate—a reflection of the severity of many of the cerebral lesions presented by the series as a whole. On clinical grounds this group may seem to have little in common with those more fortunate patients who are capable of benefiting from a programme of treatment in a cerebral palsy unit. Yet I believe it not unlikely that lesions of the same type may well be present in both classes of patient, though doubtless the extent and localization of the cerebral damage differ.

TABLE I.—DEATHS FROM CEREBRAL PALSY

Age at death (years)	*No. of cases*
0–9	19
10–19	14
20–29	6
30–54	6
	45

TABLE II.—MALFORMATIONS ASSOCIATED WITH CEREBRAL PALSY

Polymicrogyria	6
Porencephaly	2
Cerebellar atrophy (granular layer type)	2
Tuberose sclerosis	1
Sturge-Weber syndrome	1
Malformation of caudate nuclei	1
	13

The malformations comprised 13 cases or roughly 30% of the group (Table II). Polymicrogyria was found in 6 brains and was also present to a less extent in the 2 cases of porencephaly. It seems advisable to denote by "polymicrogyria" the special prenatal cortical anomaly referred to here since the term "microgyria" has been so loosely used in the past. Crome (1952*a*) has recently given an excellent account of the condition but perhaps a brief description of its main features may not be out of place in view of the fact that it is still sometimes confused with atrophic lobar sclerosis. The coarse aspect of polymicrogyria is readily identified by the finely wrinkled or bossed surface of the rather wide convolutions often found in these brains (Fig. 1), an appearance aptly likened to a chestnut kernel by Greenfield and Wolfsohn (1935). On section the reason for the name becomes apparent—one sees a wealth of small plications formed by bands of nerve cells lying deep to the surface of the brain. In one well-recognized variant the primitive cortex is composed of two such layers of nerve cells separated by a conspicuous fibre lamina (Fig. 2). The deeper of the two layers of nerve cells was regarded as a heterotopic formation by Bielschowsky (1915, 1923)—an indication, therefore, that a slowing up in the migration of neuroblasts from matrix to the periphery had taken place during the early formative phase of cortical development. In the 6 cases under review polymicrogyria was associated with symmetrical spastic paresis in three instances, with triplegia once and with hemiplegia twice. In the hemiplegias the malformation was strictly unilateral and the pyramidal tract of the affected hemisphere absent or greatly reduced in size. From the clinical standpoint small-headedness is by no means the rule—indeed 2 of the bilateral cases were complicated by a mild internal hydrocephalus. Congenital dislocation of the hips was recorded in one of these

cases and in another the presence of a pre-natal malformation had been suggested during life by bilateral coloboma of the iris. The parents of this child were first cousins but I can find no suggestion of genetic causation in the literature of the condition.

The remaining 32 cases in which no malformation of prenatal origin was found have been classified according to the main site and character of the lesions (Table III). Destructive processes

TABLE III.—PREDOMINANT SITE AND TYPE OF LESIONS IN PARANATAL CEREBRAL PALSY

Cerebral cortex (i) Cystic degeneration	13
(ii) Sclerotic atrophy	12
Lesions of corpus striatum and/or thalamus ..	5
Cerebellar atrophy (Purkinje cell type)	1
Thrombosis of sagittal sinus (recent)	1
	32

affecting the cerebral cortex and subjacent white matter predominate. In the absence of clear-cut signs of malformation pathological criteria alone are usually insufficient to allow one to determine with confidence whether the damage to the brain has occurred during the later stages of pregnancy, during birth or in early infancy. Every case has to be interpreted in the light of a good obstetrical or neonatal history—hence the advantage of examining brains of younger patients. The histories of 13 cases were sufficiently detailed to make the diagnosis of birth injury highly probable. In 8 others the pathological findings were similarly suggestive of anoxic or vascular damage but the birth histories were either not known or not suggestive of trauma. It must be realized, however, that serious birth injury may take place in apparently normal labour (Brouwer, 1949). In 3 additional cases, all examples of extensive cystic degeneration of the brain, the obstetrical histories suggested that anoxia during late pregnancy had played a contributory or even a decisive part in the pathogenesis. In one case the mother had been grossly anæmic and had suffered from transfusion rigors after which the fœtal movements ceased. In another there had been a retroplacental bleeding of considerable size while in the third case the survivor was one of twins, the other having been dead for several days at the time of birth. Such possibilities of intra-uterine anoxia should certainly be borne in mind in cases of presumed birth injury and made the subject of special enquiry. From the point of view of ætiology it is interesting to note that of the twelve patients in this paranatal group whose birth weights were known no fewer than six were premature in the sense of having weighed 5½ lb. or less at birth, while 15 out of 22 were first-born. These findings indicate that cerebral palsies are, in a substantial proportion of instances, birth palsies, to use Gower's expression, and that even in a severely mentally defective group of patients such as this malformation plays a less important role.

Little's (1862) theory that neonatal asphyxia is an important cause of cerebral palsy has recently been greatly expanded by Courville (1950) who sees in cerebral anoxia a probable pathogenetic factor common to a wide range of neonatal nervous disorders. Courville would also restrict the term "birth injury" to gross lacerations of the brain and its coverings or blood vessels. I believe it less pedantic to use the term birth injury in a wider sense so as to include all forms of cerebral damage sustained during birth. These are now known to vary from a patchy or laminar loss of nerve cells to massive softening of the greater part of both hemispheres. The strains and stresses of parturition obviously entail risks other than those of neonatal asphyxia. Apart from direct injury to blood vessels causing hæmorrhage or thrombosis there are the dangers of venous stasis as emphasized by Schwartz (1924, 1927) and before him by Holland (1920, 1922). There is the allied and highly important factor of œdema of the brain set up by serous transudates from vessels damaged by anoxia (Hallervorden, 1939). There is also the possibility that the nerves supplying the walls of blood vessels may themselves be injured at birth so that a later and perhaps progressive degeneration of tissues may result from functional circulatory disturbances (Wohlwill, 1936). Finally, there is Rydberg's (1932) theory that cerebral birth lesions commonly follow a drop in arterial blood pressure below a critical point, this failure to maintain an efficient circulation being due to extreme pressure upon the fœtal skull or to sudden and violent fluctuations in intracranial tension. The following example of cystic degeneration of the brain may perhaps be best explained along the lines of Rydberg's hypothesis:

A first-born, male infant, was delivered in a Liverpool Maternity Hospital after a labour lasting 18½ hours with a second stage of 2 hours 10 minutes. The mother had some contraction of the pelvic brim and trial labour rather than Cæsarean section had been decided upon since the baby was judged not to be large. Delivery was normal and the birth-weight 6 lb. 6 oz. The infant was limp at birth but responded rapidly to having the air passages cleared and after the administration of oxygen and carbon dioxide. Six hours later twitching of the feet was noted. The baby was feeble and reluctant to feed and five days after birth became semi-comatose and was found to be in a state of flexor spasm. The pædiatrician who saw him at this time considered that severe intracranial trauma had occurred. When admitted to the Colony at the age of 4½ years he was an idiot with spastic quadriplegia and epilepsy and death occurred two years later. The brain weighed only 507 grammes and showed extensive bilateral softening of the cortex and subcortical white matter mainly within the distribution of the middle cerebral arteries (Fig. 3).

There is, however, an important group of cases in which sclerotic atrophy of convolutions is found bilaterally in the neighbourhood of the sagittal sinus and in these it is plausible to seek the origin of the lesions in some obstruction to the venous return—as in Schwartz's theory of venous stasis. In 5 of my cases which were characterized clinically by cerebral blindness both calcarine areas and the adjoining superior parietal lobules were affected by ulegyria. Such shrunken and sclerotic convolutions almost invariably exhibit greater damage in the parts hidden below the surface of the brain (Fig. 4) and this is also true of the cerebellar ulegyria sometimes caused by birth injury.

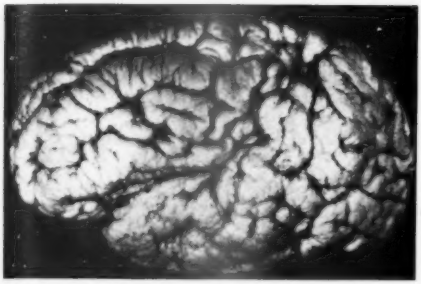


FIG. 1.—Polymicrogyria. Typical "chestnut kernel" brain.

FIG. 2.—Microscopical appearance of polymicrogyria. Note the multiplicity of small convolutions and the great reduction in the number of sulci; also the two layers of cortical nerve cells on the right. (Cresyl violet × 2·5.)

FIG. 3.—Cystic degeneration of the cerebral cortex and subcortical white matter. (Kultschitsky-Pal stain. Natural size.)

FIG. 4.—Typical ulegyria. Note the greater destruction of the deeper parts of the gyri. (Heidenhain's stain for myelin × 3·7.)

Hallervorden (1939) has attributed this special vulnerability of the gyral wall to the mechanical effect of compression of the cortex against the skull. Blood will tend to be driven into the vessels of the sulci and the ill-effects of vascular congestion and stasis upon the nerve cells may be aggravated by transudation of œdema fluid which will interfere still more with oxygenation. Such a waterlogging of the tissues is also thought to be especially likely to involve the relatively loosely constituted third layer of the cortex and to be an important factor in the production of laminar atrophy.

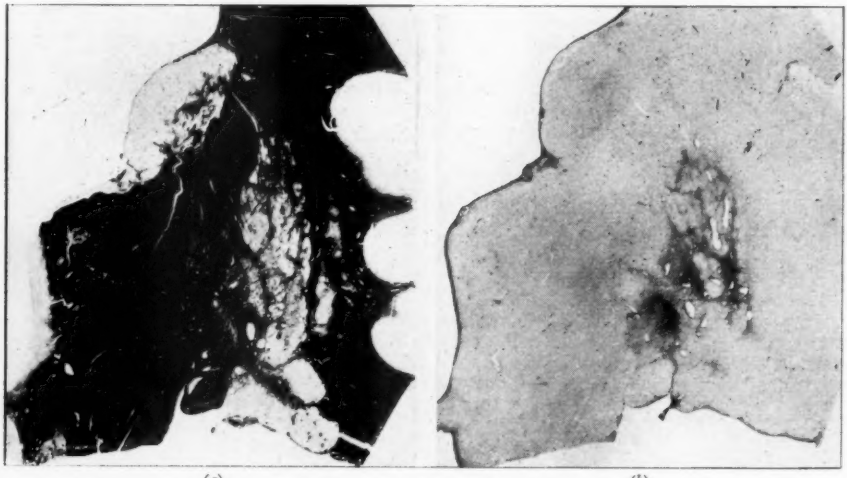
Another lesion characteristic of birth injury—and one peculiarly damaging to the growing brain—is the paraventricular softening of the central white matter which was so well described by Schwartz

(1924) and related by him to bleedings from or stasis in the radicles of the internal cerebral veins (Fig. 5). Spongy softenings or actual cavities may be found along almost the whole length of the centrum semiovale, from frontal to occipital poles. The cortex is thus undercut and the pyramidal tracts destroyed or inhibited in their post-natal growth. In certain cases, this type of lesion expresses itself in a thinning of the white matter with wide dilatation of the lateral ventricle without there being much appreciable atrophy of the cortex itself to account for this marked degree of hydrocephalus *ex vacuo*.

FIG. 5. — Coronal section of a hemisphere showing long-standing paraventricular softening.

Any discussion of the subcortical lesions of birth injury would be incomplete without some reference to *état marbré* of the corpus striatum—the commonest pathological finding in athetosis. The consensus of opinion, including the Vogts' (1951), now favours the view that this condition is not a prenatal malformation but the consequence of vascular or anoxic lesions occurring at birth or less often in early post-natal life. The abnormal networks of myelinated fibres are usually seen in regions showing conspicuous nerve cell loss and gliosis (Fig. 6) and similar evidence of a destructive process is often to be seen elsewhere in the basal ganglia or cerebral cortex of the same brain. In all probability the athetoid postures often assumed by the hand in young hemiplegics are the consequence of coincidental involvement of the corpus striatum of the damaged side of the brain.

In contrast to the predominantly neostriatal distribution of lesions in *état marbré* the late neuro-pathological sequelæ of kernicterus are seen typically in the subthalamic nucleus and globus pallidus (Fig. 7), structures seldom severely affected by birth trauma. In the only survivor of hæmolytic disease due to rhesus incompatibility recorded in this series—a child of 17 months with severe athetosis and head retraction—I found the globus pallidus, subthalamic nucleus and Ammon's horn of both sides selectively and symmetrically damaged.



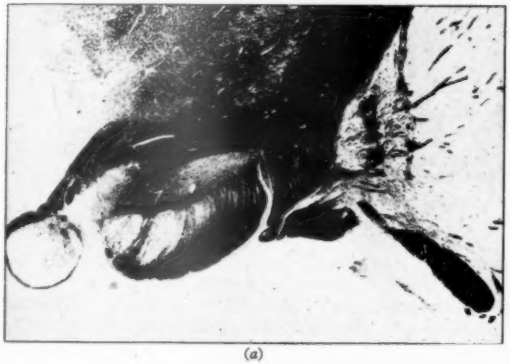
(*a*) (*b*)

FIG. 6.—*État marbré*. Note in (*a*) the abnormal myelinated fibres in putamen and caudate nucleus. The lateral part of the globus pallidus shows slight myelin loss. (Kultschitsky-Pal × 1·9.) In (*b*) the corresponding neuroglial overgrowth is seen. Note the extensive involvement of the globus pallidus. (Holzer stain × 1·9.)

In my small series of 9 cerebral palsies of post-natal origin 8 were hemiplegics, 6 being examples of acute hemiplegia ushered in by a succession of fits in a hitherto apparently healthy infant. The pathological features of these brains included false porencephaly, atrophic lobar sclerosis and hemiatrophy of the brain with intact pyramidal tracts. In all these well-known conditions vascular lesions were probably responsible for the initial damage, as in the following case:

A 9-year-old idiot previously without signs of spasticity or epilepsy suffered from mild fever and diarrhœa for two days and then had a succession of major fits involving the right side of the body. Although the convulsions were arrested by treatment she remained semi-comatose and died twenty hours later. Six hours before death she exhibited signs of right-sided hemiparesis with increased tone and exaggerated tendon reflexes in upper and lower limbs, a positive Babinski response and tonic innervation of the right hand. At post-mortem the brain showed thrombosis of the middle part of the sagittal sinus and also of both rolandic veins, the left one being the more extensively affected. There was a widespread hæmorrhagic infarction of the left hemisphere with a minimal bleeding on the right side.

This case is of topical interest, in view of Mitchell's (1952) paper on hemiplegia following venous thrombosis and also the observations on the association of gastro-enteritis and cerebral palsy made by Crome (1952*b*) and by Schlesinger and Welch (1952).



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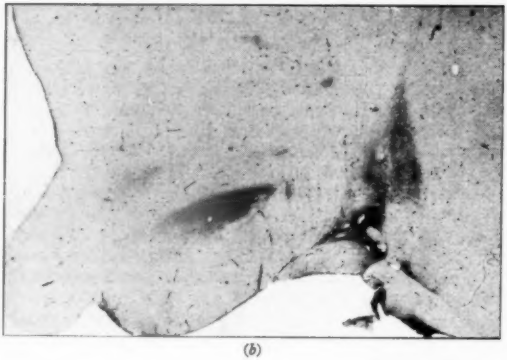

(*b*)

FIG. 7.—Late sequelæ of kernicterus. Note in (*a*) the loss of myelinated fibres in the subthalamic nucleus and globus pallidus (Kultschitsky-Pal × 3) and in (*b*) the corresponding increase in neuroglial fibres in the affected nuclei (Holzer × 3).

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Dr. P. H. Sandifer:

Incidence of Infantile Cerebral Palsy

Although it was in this country, as long ago as 1843, that spastic and athetoid infantile palsies were first described by W. J. Little (*see* Little, 1853 and 1862), yet it was in the U.S.A., in recent times, that there began a renaissance of interest in the treatment of these disorders, largely due to the work of Phelps (1940) and Carlson (1941).

In the United States Phelps' figures are generally quoted (1946). He estimates that in every 100,000 of population there will be produced, each year, 7 cases of I.C.P. whose destiny is as follows:

TABLE I

1 case	14%	Death before aged 6 (usually in infancy)
2 cases	28%	Feeble-minded Permanent custodial care needed
1 case	14%	Severe physical handicap Custodial care and education needed
2 cases	28%	Moderate physical handicap Education and treatment needed
1 case	14%	Mild physical handicap No special problem

The chief problem, according to Phelps, is the 28% moderately handicapped cases who number, as 20-year-olds or under, 400 per million of population.

In 1948 the British Council for the Welfare of Spastics (B.C.W.S.) conducted a survey in which Local Authorities in England and Wales were questioned, two-thirds replying.

The over-all average incidence rate was 0·92 per thousand child population under 16. The average incidence rates derived from each individual Authority gave 1·12 per thousand.

A further series of surveys in selected areas, conducted in this country by the Ministry of Education in 1947–48, showed wide variation in the incidence of cerebral palsy in children of school age. The highest incidence was 2·5 and the lowest 0·3 per thousand in the county boroughs providing the statistics. (In contrast, American figures show a remarkable uniformity of incidence.) From the figures provided by their survey the following tentative conclusions can be drawn:

(i) On the average the incidence of I.C.P. is a little over 1·0 per thousand children of school age.

(ii) The total number of children under 16 with I.C.P. in England and Wales is approximately 6,000.

It would be interesting to know what proportion of these 6,000 children fall into the several syndromes of infantile cerebral palsy. But figures published vary enormously due, it would seem, to varying criteria of diagnosis, particularly in distinguishing spastic from athetoid forms. Those who believe (Phelps, 1941*a*, *b*; Evans, E. S., 1946; Evans, P. R., 1948) that the athetoid are about as common as the spastic forms, stress that "tension athetosis" is frequently mistaken for spasticity of pyramidal type.

It is also important to know what proportion of these 6,000 or so children are capable of some kind of suitable training. In October 1952 a memorandum on the training of teachers of cerebral palsy children was submitted to the Ministry of Education's National Advisory Council (on the training and supply of teachers by the B.C.W.S.). This memorandum made the following estimate:

TABLE II

Capable of attending ordinary schools	25% approx.
Probably capable of benefiting from special schools	60% approx.
Incapable of benefiting from any schools because of serious mental defect (at best only suitable for training in Occupational Centres)	15% approx.

The "New Treatment" of I.C.P.

These figures give a rough idea of the size of the problem and they prompt the question whether it is a good investment to deal with it by spending large amounts of money and man-hours of skilled labour in organizing centres for the treatment of these cases.

Before venturing an answer it would seem wise to examine this "New Treatment" of infantile cerebral palsy. To me it seems to be no more than the application of the principles which underlie skill-learning in normal subjects, but with particular patience and understanding. For this reason it would perhaps be worth while to consider the process of acquiring skills.

James Collier, in his Savill Memorial Oration of 1929, speaking of localization of function in the nervous system, had this to say: ". . . we must keep carefully in mind that the function of movement and the function of sensibility are not two separate functions, but that they are so intricately blended as to be inseparable. We have no consciousness of movement in ourselves except from the sensations which result in its accomplishment. We learn every skilled act by gradually laying down, by repetition,

the pattern of the sensations which are produced by its correct performance. We reproduce them from the memory of these sensory patterns. We become clumsy with our skilled acts when out of practice because the memory of the finer details of the sensory pattern tends to fade and we reinforce them again by further repetition."

These remarks of Collier seem to me apposite to the clinical problem presented by I.C.P. For here there is more than just a motor deficit. Thus the inability to perform intended motor actions prevents the accumulation of a store of these memories of sensory patterns of movements and postures upon which voluntary movement depends. Lack of practice means lack of skill—and the untreated I.C.P. is grossly handicapped by lack of opportunity for practice.

Collier continued with these words: "There is in the function of movement a running analysis from the proprioceptive system and sometimes, in addition, from the visual and auditory systems, which is absolutely essential to this function."

The importance of this "running analysis" on motor function he illustrated by the experiments of Osborne and Kilvington on the dog. These workers connected the proximal end of the middle cord of the brachial plexus on one side to the distal end of the middle cord of the brachial plexus on the other side. The dog recovered well and could walk, run and hold down a bone with either fore-paw. Examination after recovery showed that the extensor afferents and efferents of one fore-limb had been thrown into the middle cord of the opposite brachial plexus and therefore into the hemisphere of the same side. Electrical stimulation showed that the cortical points for extension of the right fore-limb had been shifted from the left hemisphere to the right.

Experiments of this kind, and also clinical observation, as Walshe (1948) has argued, are not compatible with the view that movements are represented in fixed points in the cortex. Further knowledge of cerebral localization has followed the study of hemispherectomized subjects. This operation, first performed by Dandy and more recently by Krynauw (1950) is now almost a commonplace operation amongst neurosurgeons who seek to relieve the fits and behaviour disorders of one type of I.C.P.—infantile hemiplegia. We have learned how surprisingly well these hemispherectomized subjects can use the contralateral limbs—sometimes with less spasticity than before operation. Goody and McKissock (1951) have sought to explain this amazing fact and have suggested that, at birth, the infant has no absolute localization of function in its cerebral cortex. As they themselves put it, "the brain stands ready for training". If one hemisphere is damaged, other parts of the brain to some extent take over the function of the parts that cannot develop. Whether these functions are taken over by lower centres of the same side, cortical or subcortical centres of the opposite side or by a combination of both, it is not known. But it would seem that, in young children, this "plasticity" of the brain in re-allocating functional sites may enable the infantile diplegic and quadriplegic to do what the infantile hemiplegic does—namely to achieve some degree of motor skill by enlarging the scope of surviving brain tissue—provided there are favourable circumstances. The essence of the "New Treatment" is to provide these favourable circumstances. As Collier pointed out regarding the dog experiment of Osborne and Kilvington, functional recovery of the afferents is of enormous importance and must precede the regaining of efficient motor function which depends upon proprioceptive guidance. So, too, with learning skills by the I.C.P. patient (as in normal subjects) it is what goes in via the afferents which largely determines what comes out via the efferents. The favourable circumstance which treatment should aim at is the provision of an abundance of co-ordinated afferent stimuli, particularly from the paralysed parts, so that the child can acquire a repertoire of movement and posture memories and, through repeated practice, skill in translating them from thought into action. By way of example, in teaching the child to feed itself or to write, the hand is held, grasping the spoon or pencil, and the arm moved passively so that food is transferred from the plate to the mouth or symbols made to appear on the paper. These passive feeding and writing movements are made under guidance and control and, by repetition, a store of memories of these movement patterns is built up—memories of movement which must precede the performance of voluntary movement. It is likely, too, that the size and functional efficiency of the nerve cells concerned in the motor acts imperfectly performed in infantile cerebral palsy are favourably influenced by repeated activity.

In the treatment of infantile cerebral palsy, as in the treatment of poliomyelitis, there is the primary benefit which is the gaining of motor skills: there is also the secondary benefit of avoiding those evil complications of the disease in the form of deformity and contracture. C. D. Agassiz (personal communication, 1952) maintains that deformity and contracture are preventable in children of normal mentality taken early enough. If this challenging contention proves true, then the tenotome, the instrument which Little introduced into this country, is unnecessary. It would follow that, if more effort were expended in preventing these complications, there would be less need to try and cure them by corrective operations, with all the waste of ward accommodation and skilled man-hours which avoidable operations involve.

But such benefits, when physical disability is severe, are probably best achieved in special units; and about these we have asked the question, "are they a good investment?" I think the answer depends on the selective machinery of such units. If only those patients are admitted in whom a successful response to treatment in a reasonable period is predicted with fair accuracy, then they would seem

to be worth while. Cases which should not be (and are not) admitted are those amenable to indirect treatment—the 25% who will reach ordinary schools and who can be adequately treated in out-patient clinics by instructing the mother in what she should do. Likewise those should be rejected who are too defective mentally to benefit.

These Special Centres have shown us how surprisingly well some of their cases do. But nobody has compared, as far as I know, a treated with an untreated group. It would seem important, however, to avoid those dangers which followed the introduction of the "New Treatment" of poliomyelitis—extravagant claims for particular techniques and quaint physiological dogmas.

Adult Cases of I.C.P.

But the good results of good treatment are largely wasted if ways and means are lacking in finding these people work in later life. There are no figures available concerning the employment of I.C.P.s. The M.R.C. Memorandum No. 28 on "The Employment Problem of Disabled Youth in Glasgow" (H.M.S.O., 1952) states "The severely paralysed, no matter how intelligent or capable they may be, do not stand much chance of finding a job in Glasgow. As they themselves say: 'People take one look at you and that's the end of any chance of a job.'" The experience of B.C.W.S. suggests that this is true of the whole country.

It would be useful to know what proportion of I.C.P.s surviving infancy survive into adult life where they raise this problem of employment, also the problem of custodial care. No figures are available. The B.C.W.S. has information concerning a little over 2,000 cases. The vast majority of these are young children. The Midland Spastic Association, which is trying to register all I.C.P.s in their area, likewise find that much the greater proportion are children.

This lack of information about adult cerebral palsies has led to speculations concerning the early death of these subjects.

On going through cases of I.C.P. who came to the Royal National Orthopædic Hospital over a two-year period, 182 were identified. Of these about 79% were under 30 and 21% over 30 years old. One would expect a higher proportion of children than adults to attend a hospital for treatment and the age distribution does not show any surprising preponderance in favour of the children. The figures were as follows:

TABLE III

Ages ..	1–9	10–19	20–29	30–39	40–49	50 and over		Cases	%
Cases	49	62	32	23	9	7	Over 30	39	21 (approx.)
% ..	26·9	34·1	17·6	12·6	4·9	3·8	Under 30	143	79 (approx.)

Some Clinical Aspects of I.C.P.

My experience of this condition in infants and very young children is very limited, but at the other end of the scale I have seen the diagnosis of infantile cerebral palsy delayed till the autumn of life. These cases present as mild spastic paraparesis of late onset with pyramidal but no sensory signs. Careful enquiry reveals a life-long minor disability which has been accepted for so long that it is scarcely recognized (as may be the case with victims of mild peroneal muscular atrophy). The past history may contain information which reveals that disability is nothing new: for example, that "irons" needed to be worn in childhood; that there was delay in learning to walk; that although football was played at school, running was so clumsy that he always played in goal. With advancing years we become slower and less nimble on the legs and tire more readily. If there is any underlying motor handicap, this process will be accentuated and disability previously accepted because familiar, will now be recognized so that medical advice is sought.

In children too, one sees, from time to time, the complications of spastic diplegia—deformity—bring them before the doctor, the underlying cerebral lesion being missed. Thus a child, reported to have played games at school, may start to walk on the toes and to show an ungainly gait. Examination reveals extensor plantar responses and contracture of the Achilles tendons, in the absence of sensory or sphincter disturbance and with brisk abdominal reflexes. Again careful enquiry may provide the clue, with hints that minor motor disability has long preceded the onset of deformity.

Sometimes, in young people with spastic diplegia, the diagnosis comes up for review, because the disability is progressive. Thus a child may gain fair but delayed skill in walking, only to lose this skill later. The deterioration, in such cases, is not due to any progressive disorder in the nervous system but to a complication of that stationary disorder in the form of contracture. As the tendon shortens so walking deteriorates and disability increases.

It would seem important to use the Special Units for Infantile Cerebral Palsy for collecting information. I think long-term comparisons should be made between comparable groups treated in different ways—for example, those treated in Special Centres with those receiving no special treatment.

It is also important to test the contention of Agassiz—that deformities are preventable if children of normal intelligence start treatment early enough. If the future proves him right, we shall have gained a valuable fragment of knowledge for use in treating infantile cerebral palsy.

Prevention.—The infant brain may, we know, be damaged in various ways in different sites in the pre-, para- and post-natal periods, producing cerebral palsy of symmetrical or asymmetrical type by lesions most frequently involving the cortico-spinal tracts, the corpus striatum or both. We need to make further progress before we can do much more than think in terms of preventing cerebral palsy.

The incidence of premature births, difficult labour and neonatal asphyxia is higher for cerebral palsied than for normal infants. There is a relationship, though not always a direct one, between abnormal birth and abnormal brain. Nevertheless mechanical trauma and asphyxia sometimes do cause infantile cerebral palsy and to some extent these causes are preventable.

That there is a relationship between maternal ill-health and fœtal deformity is suggested by experimental findings in pregnant animals subjected to such insults as vitamin lack (Warkany and Schraffenberger, 1944; Warkany, 1947; Wilson and Warkany, 1950), dye injections (Gillman *et al.*, 1948), radiation (Kaven, 1938) and anoxia (Ingalls, 1950). Even with anencephalus, in which evidence has accumulated indicating a genetic origin (Schade, 1939; Böök and Rayner, 1950), it has been shown by McKeown and Record (1951) that there is a curious seasonal incidence of births with this abnormality. Why it should be that there is a higher incidence of anencephalic malformations amongst embryos conceived in March or thereabouts, is not known. But it does suggest the operation of an environmental factor in ætiology and thus provides hope that further knowledge may give some measure of control over this and other types of congenital cerebral abnormality.

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Mr. E. Stanley Evans: There are now in this country a number of cerebral palsy centres, including the pioneer centre at Carshalton, and others in Croydon, Birmingham, Edinburgh and Ivybridge. These are all special centres dealing in varying ways with the diagnosis and assessment of those suffering from cerebral palsy, and with their treatment, training and education.

The care and treatment of the spastic child is not the prerogative of the neurologist, pædiatrician, psychiatrist, the expert in physical medicine, or the orthopædic surgeon, but demands team work and participation by all these and others such as the aurist (to exclude pitch deafness), so that the child can be treated comprehensively by the physiotherapist, occupational and speech therapists, and by the expert in vocational guidance and training. In no condition is it more necessary for each specialist and therapist to play his part, not by an individual or isolated approach, but as a member of a well co-ordinated team.

The first essential is the accurate diagnosis and assessment of the physical condition of the child, as to the nature and extent of the condition, whether spastic, athetoid or ataxic, and whether mono-, hemi- or quadriplegic. In particular, the differentiation between the child suffering from spastic quadriplegia, whose muscles exhibit the spastic stretch reflex, and athetoid quadriplegia with tension, may not be easy until after a period of observation. Secondly a mental assessment must be made, so that the child who is intelligent and educable may be selected for comprehensive treatment. This may be extremely difficult even for the expert educational psychologist, and is concerned with potential mental ability and not with the ability to carry out certain performance tests demanding neuromuscular co-ordination, and the ability to communicate with others, for example by means

of speech. The degree of mental ability does not necessarily bear any relationship to the extent of the physical handicap. There are those with gross physical disability associated with widespread involvement of the brain and mental impairment for whom practically nothing can be done, others with slight physical disability and marked mental impairment, and yet others with gross quadriplegia and normal intelligence. With our present limited resources it is essential to select for treatment those who are educable and not too severely handicapped physically.

The aims of treatment are:

(1) To train the child in the elementary skills of daily living, including dressing, feeding, personal hygiene and walking, so that he may become socially sufficient and presentable.

(2) To establish and improve the various means of communication by speech, writing and typing.

(3) To educate the child and train the individual vocationally to the highest level of achievement.

These aims are more difficult to attain in the older child, but when carefully selected even these may respond in a remarkable manner if stress is laid on education, training and welfare.

Treatment is essentially conservative in character, for even when operative procedures are necessary they are but incidents in a comprehensive and prolonged programme of treatment. Physiotherapy has an important role to play and is concerned with function rather than anatomical improvement, with the conservation and development of muscle power rather than with weakening of apparently over-active muscles, and it must be physiological and dynamic rather than corrective and static. Relaxation and the elimination of purposeless movements must govern the treatment of the athetoid, whilst muscle training and the establishment of increased joint mobility is necessary for the spastic child. Exercises in the prone position, crawling and the development of reciprocal movements, are practised in some centres. Relaxation is first secured in the fully recumbent position, assisted by deep breathing exercises, and then with the aid of Guthrie Smith slings, under-water exercises, and a relaxation chair. Treatment and the training of skills proceed by easy progressive stages. The skill of feeding is acquired by passive feeding in the relaxed recumbent position, and then progressively by using a special feeding table and utensils until ultimately the child can eat and drink more or less normally. The skill of walking is acquired by achieving sitting and standing balance, the use of special skis and surgical appliances, until the child is able to stand and walk alone. Improvement in the function of the hands is secured by developing the sense of touch, by handling objects in sand, by handling large wooden bricks, and then smaller ones, by open finger and brush painting, and then by writing or the use of the typewriter in order to provide an additional means of communication.

Control of the trunk and shoulder and pelvic girdle muscles must be obtained in the spastic child, and the muscle power of the weaker antagonists of spastic muscles must be developed. This is not easy to secure because contraction of the antagonist is inhibited by the stretch reflex of spastic muscle. This difficulty is found when an attempt is made to develop the gluteus medius in the presence of spastic adductors of the hip, and provides one of the indications for operation.

Operations are of value in releasing contracted structures, thereby correcting deformities; in paralysing temporarily overactive spastic muscle groups, and in enhancing function by stabilization of certain joints. Surgery must be as conservative as possible provided the desired correction is obtained, in order that the sum total of muscle power in the limb is not seriously diminished.

If the affected foot remains in a position of fixed plantar flexion when the knee is flexed to 90 degrees, the structural contracture is probably in the soleus and posterior capsule of the ankle-joint. Posterior capsulotomy and elongation of the tendo achillis should be performed, the amount of elongation being determined beforehand. When the foot can be dorsiflexed to the mid-position only when the knee is flexed to 90 degrees, and there is a fixed flexion deformity of the knee of 30 degrees or more, posterior capsulotomy of the knee combined with distal stripping of the gastrocnemius origin is necessary. If this is insufficient, tenotomy of the semitendinosus, of the tendinous portion of semimembranosus, and of the posterior fibres of the ilio-tibial band will usually suffice. The knee should be moderately corrected in plaster, or on a Thomas's knee splint, and gradually straightened during the ten or fourteen days following operation.

Soutter's muscle slide operation and subcutaneous adductor tenotomy are indicated when marked flexion adduction contracture of the hip is present. Stoffel's orthodox operation of selective nerve section with a view to producing permanent paralysis of spastic muscles is generally contra-indicated, because the total muscle power in a limb is thereby reduced considerably. On the other hand temporary paralysis produced by crushing the nerve supply to spastic muscles may be invaluable, as it allows the antagonists to be trained and developed without evoking the stretch reflex. The abductors of the hip, for example, may be strengthened after the nerve supply to the bulk of the spastic adductors has been crushed, so that when the adductors recover the gluteus medius may be well developed, and muscle balance achieved without permanent diminution of muscle power. The function of the hemiplegic hand is often improved following an arthrodesis of the wrist, provided a suitable position is selected. A temporary plaster splint may serve as a guide to the optimum position which must combine the ability to relax the fingers and grip in the flexed position, with the increased gripping power in full dorsiflexion. Generally the mid-position should be selected with the second metacarpal in line with the axis of the radius.

The important factors in the management of those suffering from the infantile cerebral palsies are:

(1) Accurate physical and mental assessment, and the selection of patients likely to respond to treatment.

(2) Comprehensive conservative treatment at the earliest age possible, and ancillary operative procedures when essential.

(3) Formal education for the child of school age, as treatment and education are complementary, and improvement is often reciprocal.

(4) Provision of more special centres for cerebral palsy, and the active co-operation of well-informed parents. These centres are expensive to establish and to maintain, and it is reasonable to consider whether the cost is justified in view of the results obtained. Provided reasonable improvement is acceptable, the value of these centres cannot be assessed on financial grounds only.

Dr. J. P. M. Tizard:

The Future of Infantile Hemiplegics

A survey of the long-term effects of cerebral palsy was begun at the Children's Medical Centre, Boston, in 1951, and I joined Dr. Bronson Crothers, previously Head of the Neurological Department in the Children's Hospital, and Dr. Edith Meyer, the psychologist, in an attempt to discover the fate of patients with infantile hemiplegia, who had received investigation as in-patients within the previous twenty years. Here, in outline, are some of the findings of this survey.

There were records of 450 cases of infantile hemiplegia and from these we selected cases of children who had been normal at birth and who had developed normally until overcome by some disaster which was unmistakably associated with the development of hemiplegia. About 150 conformed to this requirement, that is about a third of the total, the remaining two-thirds having histories suggesting that the hemiplegia was determined at birth or possibly, in some cases, before birth.

We traced 95 cases and of these examined 51. 43 were male and 52 female. 57 had a right and 38 a left hemiplegia, but cases in which the onset was sudden were equally divided between right and left.

TABLE I.—AGES OF THE 51 PATIENTS WHEN LAST SEEN

Age	Under 4 years	4–7	8–11	12–15	16–19	20–23	24–27	28+
No. of patients	4	12	12	7	5	6	2	3

There are difficulties in selecting cases in which hemiplegia has developed after birth. Hemiplegia present at birth does not usually become obvious until the infant is several months old. As can be seen from Table II we were not wholly successful in excluding the birth palsies.

TABLE II.—MODE OF ONSET OF HEMIPLEGIA

A.	Sudden onset with convulsion	39	
	(1) Ill before convulsion	28*	(Measles 4, roseola infantum 3, triple toxoid immunization 2, "colds" 4, pertussis 1, epidemic encephalitis 1, fever ? cause 6, others 7)
	(2) Well until convulsion	11†	(1 facial nævus, probably Sturge-Weber syndrome)
B.	Sudden onset without known convulsion ..	8	
	(1) Ill before hemiplegia	7	(Pertussis 1, scarlatina 1, chickenpox 1, influenza 1, fever ? cause 3)
	(2) Well until hemiplegia	1	
C.	Meningitis	7	(Pneumococcal 2, meningococcal 1, hæmophilus influenzal 1, streptococcal 1, organism unknown 2)
D.	Head injury	7	
E.	Probably dating from birth	8	
F.	Miscellaneous	26	(Mostly of gradual onset)

*2 found to have subdural hæmatomata.

†2 found to have subarachnoid hæmorrhage.

The first group consists of children who suddenly developed convulsions, and, on recovering consciousness, were found to be paralysed on one side. The majority of these were suffering from some febrile illness before the convulsion, but 11 were well and the fit came out of a clear sky. One of these had a probable Sturge-Weber syndrome, but the remaining 10 were normal children. The mode of onset of hemiplegia was so sudden in these cases as to suggest a vascular accident. There is a good deal of indirect evidence to suggest that in many of these cases there has been an occlusion of the middle cerebral artery. However, I have heard some direct evidence from three American neurosurgeons who performed carotid arteriograms in 4 cases of infantile hemiplegia shortly after

the event, showing in each case blocking of the main stem or of branches of the middle cerebral artery in the affected hemisphere.

Some of our cases had been labelled "encephalitis" but in the great majority there was little evidence to support this diagnosis. Measles encephalitis is very rarely complicated by permanent neurological sequelæ, but I suppose that the two conditions might occur coincidentally. When the C.S.F. had been examined within the first few days following the hemiplegia it was usually normal, although in a few instances it was found to contain up to 20 or 30 white cells per c.mm. However, after an interval of a week or more pleocytosis was a more common finding, and when lumbar puncture had first been carried out after such an interval, the finding of cells had sometimes led to the probably false diagnosis of encephalitis.

In the last group (F) hemiplegia had become manifest at a considerable period following that at which birth palsy is usually detected, but we could not be certain in many that there had not been some previous brain damage, possibly dating from birth. For instance, some of these children had had recurrent fits prior to the onset of the hemiplegia.

These cases were not intentionally selected in any other way. But there must probably always be some prior and unwanted selection to prevent one's claiming a series such as this as really representative. The mildest cases of hemiplegia might not find their way to the Neurological Department, nor perhaps to a hospital at all. This point is illustrated by a film of a 3-year-old boy who has a left hemiplegia dating from birth. Although the hemiplegia is quite obvious on clinical examination, his gait is not remarkable, except that the left arm swings a little less than the right when he runs. If one listens to him running one can detect the uneven sound of his footsteps.

The importance of detecting these not very obvious cases of hemiplegia is not because the motor defect is likely to prove a very serious matter, but because of its significance in relation to selective mental defect and fits.

Fig. 1 shows the age of onset in all 95 cases excepting the 8 with known birth palsy.

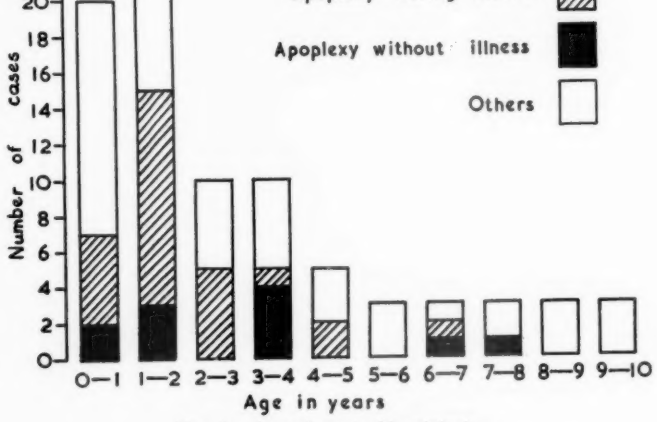

FIG. 1.—Age of onset of hemiplegia.

51 patients were seen in the Neurological Department when an interval history was obtained, particular attention being paid to motor and speech development before and after the hemiplegia; the extent of the handicap imposed by the motor defect; the effect of physiotherapy and especially of so-called "muscle training"; the time of onset and frequency and the nature of fits; behaviour and emotional problems in relation to the family situation and finally the school record and adult occupation. The patients were given physical and psychological examinations and in most cases an EEG was performed.

In few instances did the motor disorder of the affected leg impose serious handicaps. All could walk, most could run. In the great majority of cases, however, the hand on the affected side was useless for performing any fine action and, in general, these patients went through life as if they only had one hand.

Short accounts of the sensory findings have been given by Tizard and Crothers in 1952, and by Tizard in 1953.

Speech was never seriously affected except in those with gross mental defect. Even in children developing right hemiplegia after the age of 5 years aphasia lasted but a few weeks and normal speech had returned in each case by three months.

Table III gives data relating to epilepsy.

TABLE III.—EPILEPSY

Fits at onset, 32		No fits at onset, 19	
Recurrent fits from onset of hemiplegia	18	No fits subsequently	9
Fits after an interval	7	Fits after an interval	10
No fits except at onset	7		

Duration of fit-free period:

Under 1 year	1<3	3<5	5<7	7<10
3	6	4	0	4

Time interval following hemiplegia ..	1 year	3 years	5 years	10 years	15 years
Proportion of patients with recurrent fits	35%	49%	50%	66%	54%

In 32 of the 51 patients the onset of hemiplegia was accompanied by fits. Very often the first fit was generalized, and a few hours later a second fit with clearly lateralizing features occurred and, on recovery, the child was found to be paralysed on that side. In most cases fits continued; in some there was a latent period of freedom followed by fits, while in others no further fits had occurred up to the time of examination, nineteen years later in 1 case. (Some of this latter group may yet develop epilepsy; in 6 of the 16 less than four years had elapsed between the onset of hemiplegia and the time of examination.) Those who had no known fit at the onset of the hemiplegia showed the same liability to develop epilepsy later. 3 patients, who developed epilepsy after such periods of freedom, had frequent attacks for six, eight and eight years respectively, when the fits ceased completely without any change in medication. The fits were, incidentally, very resistant to medication.

In the majority of cases seizures were characterized by clonic movements of face, arm and leg on the affected side, with or without spread to the normal side and loss of consciousness. But every variety of epilepsy was encountered from momentary akinetic seizures to generalized *grand mal* attacks; the former being commoner in the younger and the latter in the older patients. The type of epilepsy not infrequently changed over a period of years. For instance, one patient had 4 generalized convulsions during measles complicated by bronchopneumonia at the age of 15 months and was left with a left hemiplegia. Nine years later she began to have attacks characterized by a feeling of numbness on the left side and jerking of the left arm and leg. These attacks occurred two or three times a week for nine years. She was then completely free from fits for a further nine years, but in the past five years has had occasional attacks of tingling of the lips and dysphagia.

That anti-social behaviour, fits of temper and violence are common in patients with infantile hemiplegia is well known and was emphasized by Strümpell (1884) in his original paper. There was a relative infrequency of such behaviour in the histories of the patients we saw. There seemed to be a rather close correlation between behaviour disorders and the frequent occurrence of fits. Not only were temper tantrums more common in children suffering from epilepsy than those free from fits, but also parents of individual children reported that meanness and tempers occurred much more often during a period of frequent seizures, while, when the child was free from attacks, his nature might appear to undergo a complete change. In thinking of these emotional storms as being connected in some way with cerebral dysrhythmia we should not overlook the great importance of environment, especially the handling of the children by parents and teachers, in the causation of behaviour disorders. Over the years Dr. Crothers and his associates acquired a profound understanding of the problems of bringing up children with mental or physical handicaps and of the emotional difficulties engendered by mismanagement. I think the wise advice given to parents was in no small measure responsible for the rather infrequent histories of behaviour disorders. To give an example, psychological tests showed again and again the characteristic intellectual pattern of brain-damaged children; good verbal abilities combined with defects in abstract reasoning and in those visuo-motor functions which apparently underlie the ability to understand symbols on which, of course, all school learning depends. Unless parents and teachers were aware of this selective mental defect, a child's superficial brightness combined with poor performance might lead to the unjust charge of idleness or inattention and therefore to rebellion on the part of a child. Another case of behaviour disorder may be found in the history of a pair of uniovular twins, one of whom had developed hemiplegia at the age of 2½ years. At 7 years of age they were quite amiable little girls but as they grew up both became disagreeable and subject to attacks of temper. The hemiplegic twin resented the fact that her sister could work in a factory and earn twice as much money as she was given for helping in the house. The normal twin, on the other hand, resented the extra care and attention given by the parents to her sister. The effect on the normal brothers and sisters of a mentally or physically handicapped child in the home would be an interesting subject for psychological study.

Dr. Edith Meyer and I tried to classify all the cases we had seen or heard of into three groups.

In Table IV the term "competitive" indicates that the patient, if a child, is able to compete socially and academically on more or less equal terms with children of his own age or, if an adult, is able to support and care for himself, albeit not necessarily at the level that might be expected from the nature of his social environment.

TABLE IV

	Competitive	Competitive with concessions	Non-competitive
Cases seen	11 (2)	11 (4)	29 (25)
Cases heard from ..	4 (3)		23 (15<22)
Dead 11			

(Figures in brackets indicate those still having seizures.)

The second group contains those patients who, as children, have had to be granted a reasonable amount of concessions by parents and teachers and as adults are able to contribute to their own needs, but who need some form of economic and social protection.

Many of the patients in the third, "non-competitive" group are already in institutions. The remainder, if children, are unable to take part in the social and academic activities of their age group, or, if adults, are entirely supported and cared for by relatives.

It will be seen from Table IV firstly, that competitiveness bears a close relation over the whole group to the incidence of epilepsy and secondly, that the prognosis for infantile hemiplegia as a whole is poor.

Sensory defects.—Sensory testing can be carried out successfully in quite young children. Mapping of the visual fields by confrontation methods and the examination of various cortical modalities of sensation, such as two-point discrimination, position sense, stereognosis, and so on, may be carried out with ease in an intelligent and co-operative child of 5 years.

We feel that our findings confirmed the observations of Penfield and Robertson (1943) on the connexion between retardation of growth and damage to the post-central cortex. All those with loss of discriminative sensation showed under-growth of the affected limbs, that is shortening as well as loss of muscle bulk. Patients with even severe motor defects who showed no shortening had no such sensory loss. I mention these sensory defects for a special purpose—to emphasize the futility of so-called "muscle training" to an arm which has no meaning to its owner. For instance, with loss of cortical sensory modalities, the patient cannot know the position of the arm in space and is presumably unable to acquire a memory of previous movements.

An intelligent young hemiplegic woman with under-growth and unilateral cortical sensory loss discussed frankly with us the medical management she had received in childhood and adolescence. With reference to that muscular training which was designed to make her in various respects independent, her most serious criticism of physiotherapy was the emphasis on training the affected arm which she had early appreciated was a waste of time. She complained of the lack of assistance she had received in trying to make the good arm doubly useful. In fact, the most useful muscle training she ever received was from a girl friend who taught her to do her hair with her one good hand.

In conclusion, I should say that I can find no clear evidence that the ultimate result is influenced by the nature of the original illness, the occurrence of fits at the onset or the side of the hemiplegia.

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Dr. N. S. Alcock: From my experience with the Dame Hannah Rogers School for Spastics at Ivybridge, I am struck with the number of unsolved problems in the clinical field: for instance, the effect of posture in controlling the involuntary movements of athetosis.

I am sure that these centres, although expensive, are justified provided they are regarded as research projects.

Dr. William Dunham: I should like to stress the fact that treatment must include constant supervision throughout the day; a succession of formal treatments alone cannot be expected to have more than transient effects on neuromuscular function. While the planning of such supervision demands expert knowledge of the difficulties in movement experienced by the individual child, its execution demands infinite patience springing from concern for his welfare. Usually, the most practical and economical solution is for the parents to work under supervision. For the intelligent child at school the co-operation of the teachers is also required. Residential accommodation providing the same essentials is necessary to deal with special difficulties of diagnosis or management.

Section of Comparative Medicine

President—C. L. OAKLEY, B.Sc., M.D., B.S., M.R.C.S., L.R.C.P.

[*February* 18, 1953]

DISCUSSION ON METALS AND SYNTHETIC MATERIALS IN RELATION TO TISSUES

Dr. E. G. C. Clarke (Department of Physiology, Royal Veterinary College):

In vitro *Aspects of Metallic Corrosion in Tissue Fluids*

A SURVEY of the vast amount of work that has been done on the use of metals in surgery shows that very few metals have been found to be satisfactory, most of them giving rise to unfavourable reactions in the surrounding tissue. Opinion is divided as to whether this is due to corrosion or toxicity. It seems probable that both factors are concerned. If a metal is completely inert, it exerts no harmful effects, but if it corrodes, the damage done is proportional to the quantity of ions passing into solution and the toxicity of those ions.

Only three metals—tantalum, vitallium and stainless steel—seem to have found general clinical application. None of these is perfect. Tantalum is scarce and expensive, vitallium is difficult to work, and not all stainless steels are inert. It seems likely that, among the very large number of alloys known, there may be some that might be much more suitable than the metals now employed. But before any clinical use could be made of these metals extensive animal experiment would be necessary, and obviously the time and expense involved in testing a large number of metals *in vivo* would be prohibitive. Before any animal experiments could be undertaken it seemed necessary to investigate the behaviour of metals in biological fluids with a view to finding a method that would pick out those most likely to be useful.

The subject of metallic corrosion in general has been extensively studied by physical chemists and it has been shown to be an electrochemical phenomenon. Unfortunately, no existing method of investigation seemed suitable for our purpose. The techniques used by many of the workers in the biological field have been chemically inadmissible. For example, current has been measured instead of potential, and when potential has been measured unsuitable reference electrodes have been employed. On the other hand, in the field of physical chemistry, the methods evolved have been for dealing with far simpler systems. One cannot take a procedure that is suitable for, say, iron in sea-water and apply it to a complex alloy in such a complicated medium as living tissue. It seemed to us desirable to start the investigation *ab initio*, with a view to finding some reproducible electrical measurement that would correlate with the known behaviour of metals in tissue. For this purpose we started with the premise that certain metals (tantalum, platinum, vitallium, gold) were inert in tissue while others (iron, magnesium) underwent gross corrosion.

Our first experiment was to measure the static potential between a plate of the metal and a calomel electrode, both immersed in serum at 37° C. This method gave no useful results, as it was impossible to obtain reproducible figures and those obtained showed no correlation with the known inertness of the metal.

We next tried electrolytic experiments, measuring the weight lost per coulomb when a current was passed between two electrodes of the metal in a bath of serum—a quantity which, for an element, would be its electrochemical equivalent. This was also unsatisfactory, but one interesting fact came

to light. In the case of the inert metals a much greater potential was needed to pass a given current through the electrolytic cells than in the case of the metals that corroded easily. It seemed that this phenomenon was worth investigating further.

The circuit shown in Fig. 1 was fitted up, current being passed through a rheostat, a milliammeter, and a cell containing serum continuously stirred. The electrodes consisted of strips or rods of the metal under test connected to a valve potentiometer. Both current and potential were noted, and the rheostat readjusted, a series of readings being obtained.

Some of the results are shown graphically in Fig. 2. It will be seen that, although the inert metals come at the top, the results are not satisfactory, as the curves cross in some cases. Thus, if we take 10 microamps as our standard current, silver comes higher than lead, but at 100 microamps the reverse is the case. A repetition of the experiment using smaller currents gave no better results, tin, for example, coming above platinum.

It must be realized that what we have been measuring is just the difference between two quantities—potential at the anode, and potential at the cathode.

In order to measure these quantities independently, the circuit shown in Fig. 3 was fitted up. It is similar to the last one, except that a calomel electrode is now included in the cell. The potential difference between the anode (or cathode) and the calomel electrode is noted for different currents. In the case of the cathodic potential, there was no correlation at all with the known inertness of the metals.

Some of the anodic potentials are shown graphically in Fig. 4. It will be seen that while the inert metals show the highest potentials the results are still not satisfactory, as in some cases again we have lines crossing, so that any figure we arrive at is dependent on the current passing.

Fig. 4 shows that while some metals such as copper and silver give straight line graphs with only a slight gradient, others such as gold, rise steeply at first and then become almost parallel with those for the former metals.

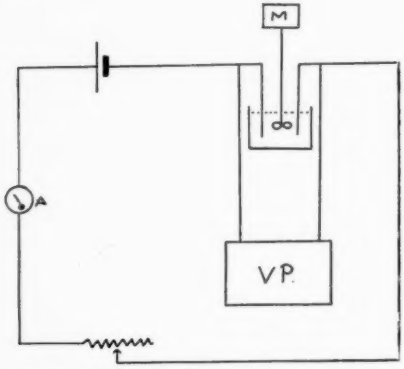

FIG. 1.—Diagram of circuit for measuring potential difference between metals in serum.

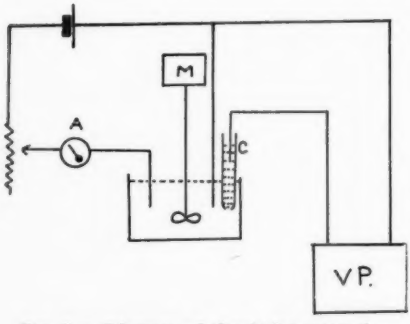

FIG. 3.—Diagram of circuit for measuring anodic and cathodic potentials.

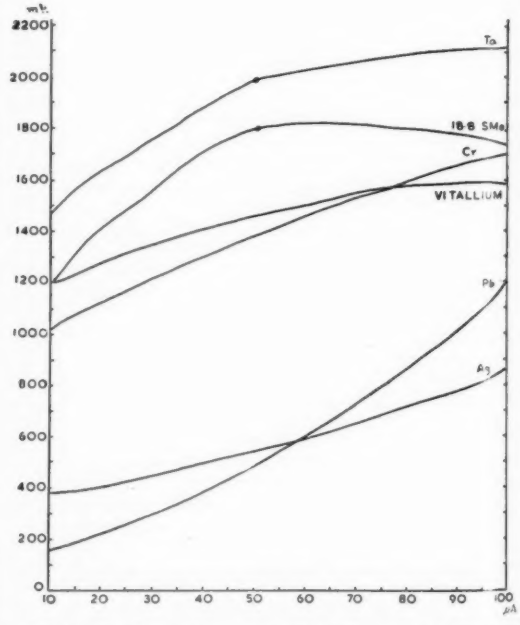

FIG. 2.—Relation between potential difference and current for various metals in serum at 37° C.

Now it must be realized that this anodic potential is the sum of two effects:

(1) The potential due to the "iR" drop across the electrolytic cell, due to the current passing through it. If the cell behaved as a simple conductor, and obeyed Ohm's law, the potential would vary as the current, and we should get a straight line graph with a slight gradient as in the case of copper.

(2) A back E.M.F. due to the reaction between the anions present and the anode. It is this potential that we want to measure. The curves for metals such as gold suggest that it increases with current finally rising to a maximum, when the graph becomes a straight line, and shows only the Ohm's law gradient.

In order to measure this back E.M.F., the circuit shown in Fig. 5 was set up. The cathode is a large calomel half cell H, connected to the bath of serum C by a KCl-agar bridge A. The anode X is formed by a strip of the metal under experiment which is rotated by the motor M to give constant stirring. Current from the potentiometer P is passed through the galvanometer G and the cell system H A C, the potential difference being read from the valve potentiometer. After a series of readings, the resistance of the cell H A C is found in the usual way by making it one arm of a bridge, using alternating current.

If this resistance R be multiplied by i, the observed current, we get V′, the potential drop due to the current. This is subtracted from V the observed potential giving us V–V′ the back potential at the anode, or anodic back E.M.F. (ABE). As V increases this value rises to a maximum where it remains constant over a considerable range. Table I shows typical results for vitallium. It will be seen that the back E.M.F. rises to about 660 millivolts.

Table II shows the values of this anodic back E.M.F. for a number of metals. The inert metals all have high positive potentials, while those that corrode readily have large negative values.

It appeared that this anodic back E.M.F. was the measurable quantity for which we had been

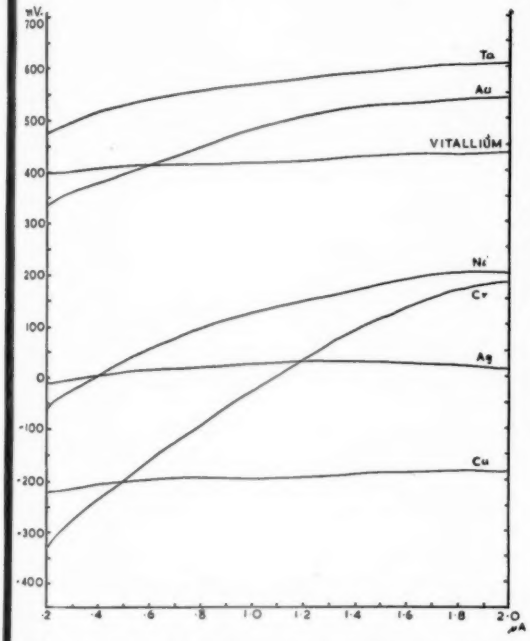

FIG. 4.—Relation between anodic potential and current for various metals in serum at 37° C.

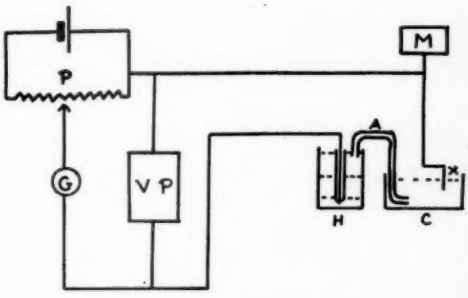

FIG. 5.—Diagram of circuit for measuring anodic back E.M.F.

TABLE I.—VITALLIUM

R = 320 ohms

V	i	V′ = iR	V-V′ (ABE)
875	0·89	285	590
1035	1·32	422	613
1215	1·82	583	632
1360	2·26	723	640
1475	2·52	806	669
1590	2·90	928	662
1682	3·19	1020	662
1767	3·46	1107	660

TABLE II.—ANODIC BACK E.M.F.

Tantalum	+ 1650
Platinum	+ 1450
Gold	+ 1000
Vitallium	+ 650
Stainless steel (18–8 Smo) ..	+ 480
Silver	+ 110
Copper	− 30
Mild steel	− 480
Zinc	− 950
Magnesium	− 1550

looking. Its great advantage is that the metals are spaced out over a wide scale (3,000 millivolts), and small variations due to experimental error, or differences between two specimens of the same metal do not affect the position of the metal in the series. We suggest that any metal high on the list, if mechanically and economically suitable, is worth testing by animal experiment.

In all, some eighty metals were examined by this method. Details of these, together with a description of a direct reading circuit for finding the anodic back E.M.F. are to be published.

Acknowledgments.—My thanks are due to Professor E. C. Amoroso for his help and encouragement in carrying out this work.

Mr. J. Hickman (Animal Health Trust, Newmarket):

Experimental Observations with Metals in the Bones of Dogs

THE experiments I propose to discuss were planned with a view to either confirming or disproving the validity of Dr. Clarke's *in vitro* work by actual *in vivo* experiments. With this object in view it was decided to implant into the long bones of dogs the following eight metals which range from those considered inert to those that should provoke a very definite tissue reaction.

1. Tantalum: Of recent years this metal has found a place in surgery and is generally considered to be inert and well tolerated by bone. For this reason and because it has an ABE of 1650 it was selected.

2. Vitallium: This chrome-cobalt-molybdenum alloy was included as a "control" because vast practical experience of its use for a great variety of orthopædic purposes during the last ten to fifteen years has shown that it causes little or no adverse tissue reaction.

3. Vinertia 'C': A British chrome-nickel-molybdenum alloy.

4. Vinertia 'S': A British chrome-cobalt-nickel alloy. These two alloys have an ABE of 880 and 750 respectively, which is higher than vitallium (ABE 650). For this reason and because they contain nickel they were considered worthy of further investigation.

5. F.M.B. steel (18–8 Smo variety).

6. F.S.T. steel (18–8 variety).

There are many varieties of stainless steels and of them the Fracture Committee of the American College of Surgeons only recommend 18–8 Smo steel for use in the body. These steels contain a percentage of molybdenum. The work of Clarke suggests that the higher the molybdenum content the more inert the steel. For this reason it was decided to test both an 18–8 Smo steel and an 18–8 steel to see if any appreciable difference in tissue reaction was detectable.

7. Silver: Although this metal has had periods of popularity, to-day it is generally considered to provoke unfavourable tissue reactions. It has an ABE of 110 and should therefore, by this classification, provoke a very definite tissue reaction.

8. Mild Steel: This steel has an ABE of − 480 and therefore should produce a very unfavourable reaction when compared with steels of the F.M.B. and F.S.T. varieties.

To test these metals, rods measuring $\frac{1}{8}$ in. × 5/32 in. were made and inserted into the femurs and tibias of dogs. Three dogs were used to evaluate each metal. Into each dog 5 rods were implanted, 3 into the femur and 2 into the tibia, one dog from each group being sacrificed at three, six, and twelve months respectively.

The implants were inserted under normal theatre aseptic and antiseptic precautions and in order to reduce complicating factors to a minimum no antibiotics were used either locally or parenterally.

I shall discuss the changes in weight of the implants and the radiological observations.

I. Corrosion and Weight Loss of Metals

Individual implants were weighed prior to insertion and on removal. The only weight changes detected were those of the silver and mild steel implants which lost 1·37 and 1·87 mg./sq. cm. of surface area respectively. At post-mortem these implants were loose and surrounded by an area of dark discoloured bone. Individual silver implants were dulled and those of mild steel grossly corroded. Implants of all the other metals were firmly implanted and showed no clinical evidence of any bone changes. In addition, they were as bright on removal as when inserted and exhibited no evidence of corrosion.

II. Radiological

The femur and tibia of all the dogs were examined radiologically immediately after operation, periodically whilst under observation and finally at post-mortem. The radiological observations under the headings of the metals implanted are as follows:

1. Tantalum: The only reaction observable up to 367 days post operation was an increased density and thickening of the cortex around the individual implants in the femur and tibia (*see* Fig.1).

2. Vinertia 'C': The reaction observed at 410 days post operation was similar to that produced by tantalum but very much less intense. However, in two of the dogs there was a marked reaction of the cortex of the tibia with partial expulsion of one of the implants. In one dog this reaction was evident by the 45th day but had considerably subsided by the 186th day post operation.

3. Vinertia 'S': A slight thickening and increased density of the cortex developed around the implants in the femur but was not very pronounced by the 378th day post operation. However, a fairly marked reaction occurred around all tibial implants which gradually subsided and was only recognizable as a thickening of the cortex by about the 200th day post operation.

4. Vitallium: A slight thickening and increased density of the cortex was present around the implants by the 361st day post operation. In two of the dogs, by the 35th day post operation, a marked cortical reaction was present around the tibial implants but this had subsided by the 200th day post operation.

5. F.M.B. Steel: The only reaction observed was a slightly increased density and thickening of the cortex around the implants except in one dog which had partially expelled one implant from the tibia by the 30th day post operation. This was accompanied by considerable cortical reaction but had subsided by the 104th day post operation.

6. F.S.T. Steel: The only reaction observable was a very slight increase in the density of the cortex around the implants with the exception of one dog in which a marked cortical reaction of the tibia developed by the 48th day but this had considerably subsided by the 133rd day post operation (*see* Fig. 2).

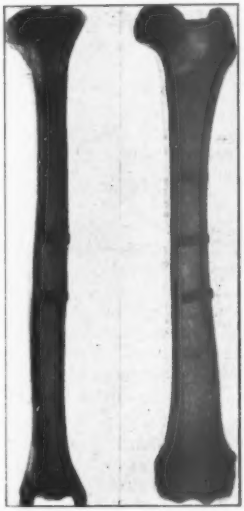

FIG. 1. — Reaction to Tantalum implants 367 days post operation. Note increased density and thickening of the cortex.

(A) (B)

FIG. 2. — Reaction to F.S.T. Steel implants (A) 48 days (B) 133 days post operation in the same dog.

FIG. 3. — Reaction to Mild Steel implants 375 days post operation. Note the area of bone destruction around individual implants.

7. Silver: An area of bone destruction occurred around the implants in the femur and was observable by the 122nd day in one dog and in another by the 195th day post operation. In addition, a thickening of the cortex with increased density developed around the implants which was very marked by the 371st day post operation. The cortical reaction around the implants in the tibia was very intense and resulted in all the implants being expelled, with the exception of one, by the 6th week post operation. This reaction gradually subsided and left a grossly thickened cortex.

8. Mild Steel: An area of bone destruction occurred around all the implants in the femur and was accompanied by a slight thickening and increased density of the cortex (*see* Fig. 3). Two of the dogs developed an intense cortical reaction around their tibial implants which gradually subsided leaving a thickened cortex with increased density.

SUMMARY

An examination of these radiographs indicates that the bone reactions to the metallic implants may be considered under two separate heads, i.e. those reactions occurring in the femur and those occurring in the tibia.

Femur.—The changes occurring in the femur are characterized by an increase in the density and thickening of the cortex around all implants, the absence of any early intense cortical reaction and the non-expulsion of any of the implants.

Tibia.—Thickening and increased density of the cortex around implants was a constant feature but, in addition, in many cases an early intense cortical reaction, often with expulsion of an implant was a common feature.

No reaction of the periosteum was observed radiologically, and preliminary histological examinations indicate that only the mild steel implants have provoked any such reaction.

In conclusion, it would appear that the weight losses together with the radiological observations indicate that Dr. Clarke's *in vitro* method does give a measure of the inertness of metals, and I would suggest that those metals and steels having an ABE above + 300 will probably provoke insufficient destructive tissue reaction to preclude their use for orthopædic purposes whereas those having an ABE below + 300 will probably provoke a degree of destruction which will make their use for orthopædic purposes undesirable.

Dr. Douglas H. Collins (Department of Pathology, University of Leeds):

Structural Changes around Metallic and Plastic Appliances in Human Bones

THIS report concerns the gross and microscopical findings in a series of human bones from persons dying at varying times after operations in which metallic or plastic appliances had been inserted. All the bones studied have been femurs after operation for cervical or pertrochanteric fracture or for osteoarthritis of the hip-joint.

Stainless-steel Smith-Petersen nails have been used in 12 patients whose bones were examined at times ranging from six days to five years after operation.

The reaction around stainless-steel screws has been studied at eleven days and at five weeks after their insertion.

Eight examples of bones containing methyl-methacrylate femoral head prostheses of the Judet type have been examined at times ranging from fourteen days to thirteen weeks after operation.

The findings in the cases of stainless-steel nails and screws have been reported in detail elsewhere (Collins, D. H., 1953, *J. Path. Bact.*, **65**, 109). In broad outline the behaviour of the bone to stainless-steel, non-corrodible nails can be summarized thus: *At five days*, repair of the injury caused by the insertion of the object has begun by organization of effused blood. *By five weeks*, the object is ensheathed by a fibrous-tissue wall. *By five months*, an incomplete shell of new coarse-fibred bone has formed in the fibrous-tissue wall. *By five years*, the bony wall has developed into a true cortex of normal lamellar bone, lined by a periosteum derived from the original fibrous track-lining. The sequestration of the foreign body is thus completed. The same type of change is seen around stainless-steel screws, except for the devitalization of compact cortical bone when a high-speed drill is used.

A comparable sequence of changes was seen in the case of the plastic prostheses when the fibrous and bony tissues surrounding the inserted stem of the appliance were studied microscopically, although no very longstanding post-operative cases have yet become available for examination.

It seems clear that, when a bland or biologically inert material, such as modern stainless steel or plastic, is inserted in the medullary cavity or spongiosa of human long bones, a succession of changes takes place in the tissues around the object, leading ultimately to its sequestration. It becomes separated and excluded from the internal structure of the bone.

A bland or biologically inert material may be defined as one which does not destroy the vitality of the adjoining tissues, which provokes no inflammatory response beyond that occasioned by the trauma accompanying its insertion and by its presence as a physical and non-vital structure, and which does not impede the process of fibrous or osteogenic repair. The bland material must itself be able to resist corrosion by the tissues and tissue fluid in which it lies. The stainless steel concerned in this series of observations comprised 18% chromium, 8% nickel, small amounts of silica and manganese and a trace of carbon, and both this material and methyl-methacrylate plastic, within the period of time covered by the observations, appear biologically inert by these standards, for there is no evidence in the human tissues studied of a granulomatous or giant-cell reaction around these buried materials or of impairment of repair processes or of interference with remodelling of the bone structure. On no occasion has there been any evidence of osteolysis in the neighbourhood of the appliances, resembling the electrolytic absorption of bone which used to be seen around the old-fashioned corroding metal nail.

It is important not to confuse resorptive changes resulting from pressure of the artificial femoral head on the amputated neck of the bone with a possible toxic influence of the material itself. Extensive changes of bone structure both destructive and degenerative may result from wholly mechanical influences.

Mr. John T. Scales (Plastics Research Unit, Institute of Orthopædics, University of London):

Tissue Reactions to Synthetic Materials

THE plastics industry has, during the last fifteen years, produced a great number of synthetic organic materials having widely differing chemical and physical properties. It is this diversity of properties which makes them of interest for surgical purposes. Because of greater mechanical strength, metals are not likely to be replaced for screws, nuts and bolts, and plates. For other purposes such as joint interpositions and the replacement of bones, the synthetic resins have considerable possibilities. To some extent the suitability of a resin for surgical purposes can be predicted with a knowledge of its chemical composition, and it is therefore desirable that manufacturers supply a complete qualitative analysis of any synthetic material which is to be used. This question of analysis raises difficulties as the disclosure of the composition of a particular material may be thought to lead to leakage of information from one company to another, especially if the work is published. All materials, therefore, should have a reference number allocated to them by the manufacturer so that if work is published about a particular resin and others wish to use the material, they will be assured of a supply which has the same composition.

Necessary Qualities of Synthetic Materials to be Used as Implants

The implant must:

1. Not be physically modified by tissue fluids.
2. Be chemically inert.
3. Not excite an inflammatory or foreign body cell response in the tissues.
4. Be non-carcinogenic.
5. Not produce a state of allergy or hypersensitivity.
6. Be capable of standing up to mechanical strains imposed upon it, as for example, friction when placed in a joint.
7. Be capable of being fabricated in the form required with reasonable ease at a relatively low cost.
8. Be capable of being sterilized.

Tissue reactions to plastics.—All plastics are complex organic compounds. In their manufacture there may be incomplete reaction of their constituents so that, when implanted in the body, various compounds may diffuse out from them. These may, or may not, excite a tissue reaction. Furthermore, many resins contain a number of additives such as catalysts, plasticizers, pigments, stabilizers, fillers, and stripping agents.

Table I shows common examples of these various agents. Certain of these may be capable of producing tissue reactions themselves, although the resin itself is inert.

TABLE I

Catalysts	*Plasticizers*	*Pigments*
Hexamethylenetetramine	Dimethyl phthalate	Mercuric oxide
Benzoyl peroxide	Dibutyl phthalate	Titanium dioxide
Mercuric chloride	Adipates and sebacates	Carbon black
Hydrochloric acid	Tricresyl phosphate	Red lead
Benzoin	Castor oil	Cadmium sulphide
Sodium hydroxide		Guanine crystals
1-Hydroxy-Cyclohexyl-Hydroperoxide-1		Iron oxide
Accelerators	*Stabilizers*	*Lubricants*
Cobalt naphthenate	Diphenylamine	Stearic acid
Iodine	Epichlorohydrin	Metallic soaps
Perbenzoic acid	Lead arsenate	Waxes
	Cadmium stearate	
Inhibitors	*Fillers*	*Stripping agents*
Hydroquinone	Alpha cellulose	Polyvinyl alcohol
Pyrogallol	Wood flour	
Sulphur	Coconut shell	
Anthracene	Keratin	
Carbon black	Kaolin	
	Asbestos	

Polymethyl methacrylate resins produced in this country are remarkably inert in the body, but in America it has been stated that some acrylics promote a tissue reaction (Wilson, 1946; Dufourmentel, 1948). The reports, however, do not give details of the composition of samples used. It may be that they contained different plasticizers or were incompletely polymerized. It has been shown by Spealman and his associates (1945) that local application of monomethyl methacrylate to the skin of rabbits produced a fleeting irritation with mild erythema at the site of application. Reapplication ten days later resulted in evidence of sensitivity in 20% of the rabbits. The completely polymerized unplasticized polymer, however, causes no irritation and is widely used for dentures and artificial eyes.

In America certain samples of polythene solvent cast by the Dupont Co. had been adulterated by the use of a stripping agent, dicetylphosphate. In contrast to other reports about polythene, Yeager and Cowley (1948) found that this particular batch of polythene provoked a marked fibrous tissue response. They have used this type of polythene for repair of hernias because of the marked fibrous tissue reaction that was induced when it was implanted in the abdominal walls of experimental animals. In this country polythene is not solvent cast and no similar reaction has been reported with the film. For certain applications, however, the use of a material containing a contaminant is advantageous providing that the reaction can be controlled.

FIG. 1.—Tissue reaction surrounding implant of phenol formaldehyde cast resin (Catalin 190M) inserted 660 days previously in anterior wall of guinea-pig. Inflammatory and giant cells—nil. Vascular reaction—nil. *Composition*: pH 6·43. Free phenol 0·31%. Free formaldehyde 0·24%. Condensation catalyst—sodium hydroxide (approx. 2%). × 82.

FIG. 2.—New bone formation around distance piece of phenol formaldehyde cast resin (Catalin 22K) inserted 168 days previously. *Composition*: pH 6·72. Free phenol 0·83%. Free formaldehyde 0·15%. Condensation catalyst—sodium hydroxide (approx. 2%).

The preliminary study of the response of the tissues to materials which may be of use in surgery is best carried out in small animals such as the rat or guinea-pig. Prolonged observation is essential. Turner (1941–42) has shown that a particular phenol formaldehyde resin produced by the Bakelite Corporation implanted in the anterior abdominal wall of rats resulted in the development of spindle cell fibrosarcomas at the site of implantation in four out of nine rats which survived for over twenty months. We have had samples of a British product, Catalin 190M, implanted in guinea-pigs for over two years with no more than minimal fibrous tissue response (Fig. 1). A similar resin, Catalin 22K, has been implanted as a distance piece in the mid-shaft of a femur of a goat (Fig. 2). X-rays show no changes in either the bone or the callus. There is a condensed layer of new bone in contact with the implant. These apparently contradictory findings may be due to species difference, or to difference of chemical composition of the resin. An idea of the toxicity of these resins can thus be obtained by animal experiments, but the mechanical effects such as friction to which a prosthesis may be subjected in the human body can rarely be gauged.

Mechanical Properties of Materials

Abrasion of the surface of the implant may produce fine particles which, because of their size and shape, will produce a tissue reaction. LeVeen and Barberio (1949) believe that besides the chemical properties of the resin, due attention should be paid to its physical characteristics. Their method of study was to inject plastics in a finely divided form, intraperitoneally. In this way a large surface

area per unit mass is brought into contact with the tissues, the particles being small and irregular in shape. The conditions required for the production of fine particles are sometimes present when implants take part in the formation of a joint.

A knowledge of the surface hardness and abrasion resistance of materials is therefore of importance. Fig. 3 shows the typical reaction induced by the implantation of a small piece of pure polythene rod in the anterior wall of a guinea-pig 774 days previously. There is a thin laminated layer of fibrous tissue forming a capsule between the implant and the loose connective tissue surrounding the muscle. Being a non-wettable and non-porous material, there is no adhesion of the capsule to the implant which was lying free in the capsular space. There are no giant cells or other inflammatory cells present. Contrast this with Fig. 4 which shows the tissue reaction induced by the same material when abraded to fine particles in a joint.

FIG. 3.—Intramuscular implant of polythene rod inserted in anterior abdominal wall of guinea-pig 774 days previously. *Capsule*: Loose fibrous tissue not adherent to implant, which was removed prior to cutting section. Inflammatory and giant cells—nil. Vascular reaction—nil. × 82. Polythene—I.C.I. (Grade 7).

Case I.—A female patient, aged 53, suffering from bilateral osteoarthritis, had a left-sided vitallium cup arthroplasty carried out, followed thirty-five days later by a right-sided polythene cup arthroplasty. Nine weeks after the operation the right hip was painful. After a further seven weeks there was increased pain with restriction of movement and an X-ray suggested that the cup had worn through. There was also evidence of erosion of the acetabular roof. It was found at operation that the cup was perforated in the weight-bearing area, and that there was a moderate amount of straw-coloured fluid in the joint as well as fibrous tissue about ½ cm. thick lining and surrounding the acetabulum. Histological examination of tissue removed from the joint showed small optically active particles of polythene associated with a foreign-body tissue reaction (Fig. 4).

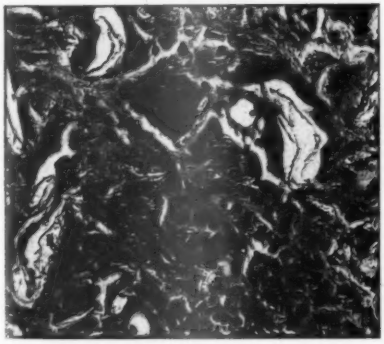

FIG. 4.—Tissue reaction induced by particles of pure polythene (Grade 7, I.C.I.) produced by abrasion of hip cup. Giant cells and profuse fibrous tissue reaction. × 84.

This case (Newman and Scales, 1951) emphasizes the importance of what one might call the "mechanical suitability" of the material. If, however, the polythene is not exposed to weight-bearing, it appears to be a most useful material for bone replacement.

Case II.—A boy, aged 12, was admitted in 1949 to the Royal National Orthopædic Hospital with fibrous dysplasia of the tibia and femur of the left leg (Fig. 5). A femoral replacement consisting of the head, neck and great trochanter and upper half of the shaft of the femur was carved from a block of polythene. It was 8 in. long and weighed 7½ oz. Channels were cut in the prosthesis for attachment of the flexors, extensors and abductors of the hip. Amputation was performed on August 2, 1949, by Mr. H. J. Seddon (Seddon and Scales, 1949), and the prosthesis inserted.

The boy made an uneventful recovery and is now fitted with a standard above-knee artificial limb. X-rays taken in March 1952 show an outline of the head of the femur which is smooth and regular (Fig. 6). His condition continues to remain satisfactory. The acetabulum was not touched at operation and, in consequence, the polythene did not come into contact with a rough surface of bone, as was the case with the polythene hip cup (Case I). Also the prosthesis is not transmitting weight, the artificial limb being ischial-bearing.

FIG. 5.—Fibrous dysplasia of left femur.

FIG. 6.—X-ray thirty-one months after operation showing prosthesis. N.B.—Outline of "femoral" head appears normal.

Porosity of materials.—If an inert material which is non-porous is implanted in soft tissues, it becomes loosely invested by a thin fibrous tissue capsule containing a small quantity of tissue fluid. Fig. 7 shows a tissue reaction induced by a pellet of Staybrite stainless steel implanted two years previously in the anterior abdominal wall of a guinea-pig. If the material is microporous, it can be invaded by fibrous tissue and become incorporated in the tissues. Fig. 8 is a transverse section of a multifilament polyvinyl chloride thread which has been completely invaded as well as encapsulated by fibrous tissue; this implant was inserted 289 days previously. There is no increased vascularity or inflammatory cell reaction. The thread was manufactured from low molecular weight polyvinyl chloride with no additives. Similarly Fig. 9 shows invasion of microporous polyvinyl chloride film.

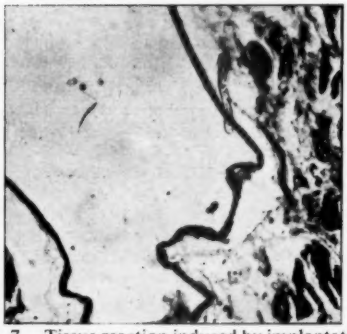

FIG. 7.—Tissue reaction induced by implantation of pellet of Staybrite stainless steel 730 days previously in anterior wall of guinea-pig. *Capsule*: Compact fibrous tissue not adherent to implant. Inflammatory and giant cells nil. Vascular reaction nil. × 82. *Composition*: Chromium 18%, nickel 8%, carbon 0·12%, manganese 0·30%, silicon 0·6%. (Staybrite F.M.B. Steel—Firth-Vickers Ltd.)

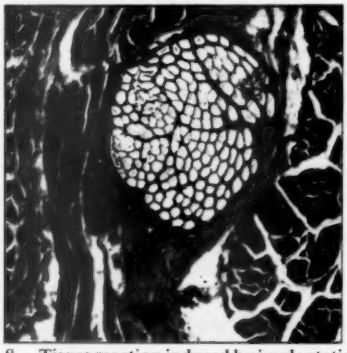

FIG. 8.—Tissue reaction induced by implantation 289 days previously of multifilament polyvinyl chloride thread in anterior wall of guinea-pig. *Capsule*: Compact fibrous tissue with concentration of nuclei adjacent to implant. The fibrous tissue has infiltrated between the filaments of the thread. There is no tissue fluid space. Inflammatory and giant cells nil. Vascular reaction nil. × 34. (Rediweld Ltd).

FIG. 9.—Tissue reaction induced by implant of porous polyvinyl chloride sheet in anterior abdominal wall of guinea-pig 56 days previously. *Capsule*: Adherent to and penetrating the surface of the implant. Inflammatory and giant cells nil. Vascular reaction nil. × 37. *Composition*: Carbon black, dibutyl phthalate 70%, wood flour 2% (Porvic—Pritchett & Gold and E.P.S. Ltd.).

FIG. 10. — Tissue reaction induced by urea formaldehyde resin containing 40% alpha-cellulose in anterior abdominal wall of guinea-pig 289 days previously. *Capsule*: Thin compact fibrous tissue not adherent to implant. Inflammatory and giant cells nil. Vascular reaction nil. × 134. Rockite urea formaldehyde 21,051/13/2. pH 6·7. Free phenol and formalin, a trace.

The use of certain fillers also appears to influence the relation of the capsule to the implant. In Fig. 10 can be seen the tissue reaction produced by an implant of urea formaldehyde containing 40% of alpha-cellulose as a filler. The implant was free in the capsule and fell out during sectioning. Fig. 11 shows how the addition of keratin (50%) to urea formaldehyde resin results in the fibrous tissue adhering to the implant. The reason for this difference is not known. Hurst, Grow, Levine, and Perlmutter (1951) have shown that a sponge of formalized polyvinyl alcohol, produced under the trade name "Ivalon" by Invano Incorporated (U.S.A.), appears to have no toxic properties or undesirable chemical reactions in mammalian tissues, and further that it becomes completely infiltrated with fibrous and collagenous tissue. Non-porous implants of polyvinyl alcohol are, we have found, encapsulated in the normal manner. This varying behaviour of the fibrous tissue capsule according to certain properties of the implant is of considerable interest. Although for some purposes it is desirable to have a surface which is not invaded by tissue, for example an articular surface, it

FIG. 11. — Tissue reaction induced by implant of urea formaldehyde resin filled with 50% keratin (bleached ground hoof) implanted in anterior wall of guinea-pig 100 days previously. Refractile particles of keratin can be seen. Fibrous tissue adherent to implant. Catalyst—benzoic acid. × 74. (Hornflowa.)

is of paramount importance to incorporate the remainder of the implant within the host tissue. In operative procedures involving the replacement of a portion of bone, it is undesirable to rely on the present method of fixation by means of intramedullary pins, nuts and bolts, or plates. Mechanically the union cannot be sound and the bolts particularly are liable to fatigue failure. It may well be that experimental work with suitable porous and/or filled materials will give results of considerable value. Callus and new bone will form around the implant of an inert synthetic material and this callus can be induced by the use of bone grafts in contact with the implant. This has been demonstrated in certain experiments which are at present in progress. The permanence of this new bone which may not be subject to normal forces remains to be established.

The trauma caused by implantation of pellets of materials in animals is usually minimal if distant incisions are made and a trochar and cannula used. The fibrous tissue formation and inflammatory cell response is directly related to the implant. In extensive surgical procedures, fibrosis normally results as a part of the process of repair and this formation, if excessive, may not therefore be

attributable to the implant. The production of fibrous tissue also may vary with the individual. The post-operative use of ACTH may possibly be of value, especially in the case of reconstructive surgery of joints where it is important that excessive granulation tissue be prevented in order to avoid limitation of movement.

Summary

Industrial synthetic materials may contain a number of additives or contaminants; a table showing some of these is given. If these resins are to be used in contact with tissues, systematic experimental work is required. We have been carrying out this type of work for more than two years, studying the reaction of tissues to large synthetic inserts in bone and investigating a wide range of materials in soft tissue in guinea-pigs. Certain materials have been used for bone replacement in the goat. (This work is being carried out with the aid of a Medical Research Council grant.) We have also been concerned with the manufacture of prostheses and their use in man. It is clear that the materials used must possess certain defined properties. The tissue reaction can be modified by the chemical constituents of the plastic, by the size of the implant and the formation of its structure. Mention is made of some orthopædic uses of these materials. Only acrylic resins and nylon monofilaments have so far been extensively used in surgery, but many other plastics are available and the judicious use of them—sometimes in combination with suitable metals—may contribute to the advancement of surgical techniques.

I would like to thank the staff of the Royal National Orthopædic Hospital and the Institute of Orthopædics, and especially Drs. C. H. Lack and H. A. Sissons of the Department of Pathology, Institute of Orthopædics, for their advice and the preparation of histological specimens, also Mr. R. J. Whitley, F.R.P.S., of the Photographic Department of the Institute of Orthopædics. My thanks are also due to Mr. S. Holt of the Plastics Division of Imperial Chemical Industries Ltd., and Mr. M. Apley of Catalin Ltd.

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Section of Radiology

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[*February* 20, 1953, *contd.*]

Treatment of the Meningeal System by Means of Radioactive Colloidal Gold and X-rays

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THE following is a brief outline of two methods of irradiation of the spinal cord and meninges which may eventually be complementary and which we have been developing at Oxford. In presenting this I do so merely as a spokesman for the department, since most of the ideas were those of Dr. Frank Ellis and a great deal of the hard work has been done by our physicist Raymond Oliver. The work is still very incomplete and we offer this only as an interim report which may be of value to others working on similar lines.

Treatment of the spinal cord by X-rays is difficult technically if a uniform dose is to be aimed at, since the cord is at a varying and sometimes considerable depth from the skin. The use of a single posterior field is far from ideal and so are attempts to combine several fields if they are conventional in shape and discontinuous.

The problem can be solved, however, if one is prepared to use fields of unconventional size and shape.

If we have two fields inclined to each other and at a constant angle to the skin, then the depth at which they will intersect below the surface will be a function of their separation. If they are inclined at 45 degrees to the surface, then they will intersect at a distance below the skin equal to half their distance apart.

If we wish to irradiate the cord it is therefore possible by simple geometry to obtain for each case two such fields which will cross fire on the cord at all its levels. These fields will present as projections of the cord and the dose at the cord will be lower where they are farthest apart than where they are closer, but correspondingly here there will be room for a direct field between them to be used to make up for this.

In treating a girl of 19 who had had an extensive ependymoma incompletely removed from the spinal canal we made up a mould on the above lines and, to minimize irradiation of the ovaries, we treated the sacral region by means of the 2 field wedge combination (Fig. 1). This has been published

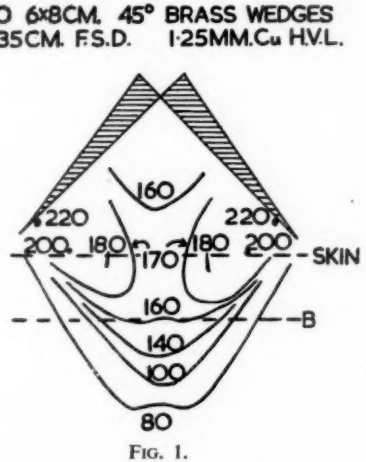

FIG. 1.

before (Ellis *et al.*, 1950). The radiation falls off very rapidly below line B and by standing the set up off the skin accordingly, we were able to get an adequate depth dose with a very rapid fall off toward the pelvis.

To speed up this lengthy process we designed a plaster shell for the patient to lie in. A Perspex plate carrying a wire grid is held in contact with the skin, and a radiograph taken with a diagnostic tube in the same relative position as it is intended to use the therapy tube. The necessary field size and shape can then be seen on the film and drawn out on the Perspex plate.

It still remains of course to determine dose rates and isodose surfaces for these fields. This needs direct measurement, but calculation has given good agreement with measured doses.

Nevertheless, however well this is done, one must be concerned about possible late damage to the cord and epiphyses, especially in the treatment of medulloblastoma in children. It would be better if irradiation could be given to possible seedling growths on the meninges in a much more localized fashion, and we set out to see if a suitable radioactive material could safely be introduced into the C.S.F. Radioactive colloidal gold seemed a suitable material immediately available, and we have done some experimental work to see if its use is feasible.

We wanted to know, apart from all questions of dosage and effective treatment depth,

(*a*) Whether the material itself would be irritant.
(*b*) Whether and how it would diffuse through the C.S.F.
(*c*) Whether it could be removed after being once introduced.
(*d*) Whether it would enter the blood stream in any appreciable quantity.

Preliminary animal experiments indicated that injection of the colloid itself into the meninges would not be likely to produce any chemical irritation, and we, in discussion with Mr. J. Pennybacker, therefore felt justified in doing some tracer studies in a patient with a cerebral tumour whose prognosis was regarded as hopeless and very short. She was actually in a terminal phase following recurrence of a glioma after previous exploration and radiotherapy.

With the co-operation of the neurosurgical department, 50 ml. of colloidal gold solution with a total activity of 480 μc. were injected into the subarachnoid space by lumbar puncture. Specimens of C.S.F. were removed from here and from the right ventricle at ¼, ½, ¾, 1, 4, and 16 hours later and counted.

Fig. 2 shows the results expressed in percentage activities relative to the injected material.

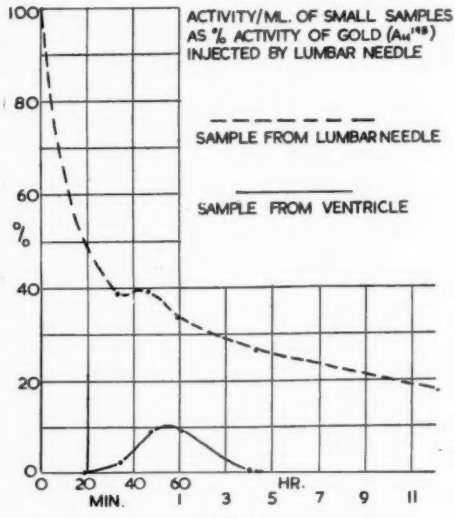

FIG. 2.

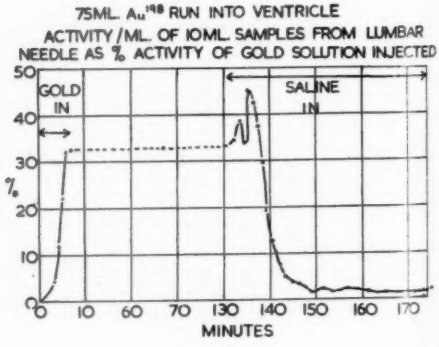

FIG. 3.

It will be seen that during the first hour fluid removed from the lumbar puncture route showed 35–40% of the initial concentration; it fell to less than half of this at 16 hours.

The activity of ventricular specimens never rose above 9% falling sharply to 0·6% at 4 hours and being 0·016% at 16 hours.

On the day following the injection, blood samples showed a concentration of only 0·026% and urine activity was 0·007% indicating that the amount of gold present in the blood could not be greater than 2·5% of the total at that time.

No alteration in the condition of the patient was observed during or subsequent to this investigation which could have been ascribed to this interference.

Obviously, however, nothing like a uniform distribution of the material had been obtained by this method.

A different technique was therefore tried. 74 ml. of the active colloid with an activity of 2 mc./ml. were run into the left ventricle of a similar patient. Simultaneously C.S.F. was allowed to flow from a lumbar puncture needle and was collected in samples not greater than 10 ml., 75 ml. in all being collected. The whole procedure took seven minutes.

Two hours later, normal saline was run into the ventricle and lumbar puncture samples collected as before. Altogether 300 ml. of fluid was collected, corresponding to 290 ml. run in.

Midway between these procedures a single lumbar puncture sample was taken together with a sample of venous blood. The samples were measured as before.

The results are as shown (Fig. 3), a rapid rise occurring during the running-in phase, and an equally rapid fall in washing out, but the latter reaching an apparent stable level.

The absolute counting of the specimens showed that 64·4% of the administered gold had been recovered from the lumbar puncture route.

The blood sample showed a negligible activity.

Four and five days later, two further samples were obtained by lumbar puncture; both these when corrected for decay showed concentrations of only 0·005 to 0·01% as opposed to 0·2 to 0·3% during the last phases of the washing out. This indicates that the gold not removed had not remained in suspension in the C.S.F.

During the washing out process, her stupor deepened; she looked shocked at the end of it and vomited a few times.

The patient died nine days after this procedure, and again both the neurosurgeons and ourselves were satisfied that the progress of the patient had not been materially altered by this interference.

At post-mortem examination, measurements with a lead collimated counter were made. Activity appeared to be confined to the C.N.S., being uniform along the cord but higher over the hemispheres.

A number of post-mortem specimens were also counted after fixation. Some slices of liver showed a definite but low activity, the spleen showed nothing above possible contamination and suprarenals, lymph node and rib samples showed no activity.

Brain slices all showed there to be little activity within the brain, the activity being localized to the serosal surfaces. The meninges in the region of the pituitary showed very high count rates and the material seemed to have entered the substance of the pituitary.

That is as far as we have gone. Obviously much more work is needed before its use can be considered as a clinical treatment. We need further and more detailed investigations of the neurological effects and to know properly the long-term effects animal experiments in more detail will be required.

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[*March* 20, 1953]

The Value of Radiology in Assessing the Progress of Duodenal Ulceration Under Treatment

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SUMMARY

THE literature on the value of radiology in assessing the progress of duodenal ulceration under treatment is reviewed. In spite of very extensive references, no paper has dealt with the specific problem postulated.

134 consecutive cases of duodenal ulcer were examined by the authors; an ulcer crater was seen with reasonable certainty in 63, all of which showed some cap deformity as well. Many of these were re-examined after intervals varying between a few weeks and two years during which they had been treated medically. In most instances the ulcer crater became smaller as pain subsided. Frequently the crater was still present though the pain had completely remitted. It is argued that while symptoms are still present, re-examination by a barium meal is unnecessary, and after symptoms have disappeared radiological "control" of treatment is not of value in the majority of cases. The reasons for these conclusions are detailed.

71 of the patients showed duodenal cap deformity only; the X-ray appearances remained unchanged in the great majority of cases regardless of the symptoms; re-examination took place after intervals of from a few weeks to three years. It is concluded that radiology has little to offer which will help the clinician in deciding how effective treatment has been or what further treatment should be instituted.

Recurrence of similar symptoms is not an indication for re-examination providing the original diagnosis is not in doubt, and that good radiographs were taken on a previous occasion and are available for inspection.

If the character of the symptoms changes, suggesting for instance obstruction or malignancy, re-examination is, of course, indicated.

Introduction

The fairly high accuracy of radiological diagnosis in the clinical syndrome of duodenal ulcer is fully established. It has been checked against the operative findings (Akerlund, 1923; Berg, 1925; Wylie, 1936) and with this side of the subject we are not concerned. The diagnosis is usually based on the clinical picture and the finding of duodenal cap deformity with or without the supposed demonstration of the ulcer crater (Chaoul, 1923; Buckstein, 1930*b*; Carman and Sutherland, 1926).

It was felt that more information was required in order to assess the value of radiological re-examination as an aid to determining the progress resulting from treatment.

Firstly, in patients with symptoms consistent with duodenal ulceration and with duodenal cap deformity, it was necessary to know how often a crater could be seen with reasonable certainty; secondly, when an ulcer crater was demonstrated, whether radiological examination would be of value in showing its disappearance under medical treatment. Finally, in cases in which no crater was proved, whether the cap deformity would alter as symptoms waxed or waned.

We hoped to find out, both from the literature and from our own observations, whether there are reliable radiological signs of healing; and, if so, whether they are of clinical value. For example, if all craters were found to diminish in size and disappear in coincidence with the diminution and disappearance of the symptom of pain, the reliability of the radiological sign would be established; but it would also be shown that re-examination by a barium meal was of no value, for it would be easier and equally reliable to judge healing by pain alone. Again, for example, if no change could be detected in the duodenal deformity of patients whose symptoms progressively improved and vanished, the radiological sign would clearly be unreliable, and, therefore, of no value.

The economic importance of this aspect of the problem is shown by the fact that approximately 650 barium meals were carried out in one year at St. Bartholomew's Hospital for the assessment of progress of duodenal ulcers under treatment. Of these 134 came to us personally and form the material of this study.

Review of Literature

(1) *On the Frequency of Radiological Demonstration of the Ulcer Crater*

In our attempt to discover the value of any radiological signs of healing, we wish to find the proportion of patients with radiological evidence of duodenal ulceration who exhibit definite ulcer craters. This is because those with and those without a crater visible on the radiographs fall into different classes as far as the radiological signs are concerned; and it may be that we shall be able to help the physician in one class but not in the other.

Leaving aside those who have not relied upon the compression technique for their diagnosis, and speaking generally, the published work may be divided into three classes.

(*a*) There are those authors, concerned primarily with diagnosis, who discovered the number of patients with "typical" symptoms showing ulcer craters. Some of their patients had no abnormal findings at the barium meal examination. Their results are only of secondary interest as far as this paper is concerned. Ettinger and Davis (1934) found duodenal ulcer craters in 50% of such patients. Templeton, Marcovich and Heinz (1938) found craters in 63%.

(*b*) A few authors have not specified exactly the criteria for inclusion in their series.

Clark and Geyman (1934) say "During 1932 we found positive niche evidence in 54% of all cases of duodenal ulcer".

(*c*) Only a few of the authors give us pertinent information: Akerlund (1921 and 1931) found in his first and second series that 60%, and at least 75% of his patients with radiological evidence of duodenal ulceration showed duodenal ulcer craters. From the figures of Templeton *et al.* (1938) we are able to deduce that 67% of his patients with typical symptoms at the time of examination, and some radiological evidence of duodenal ulceration, exhibited definite craters on radiological examination. Geyman (1932) showed "niches in 41% of bulbs having a deformity characteristic of duodenal ulcer", and then "in the past six months, due to improved accuracy of technique and interpretation, we have shown niches in 64%". Berg (1925) says "niches could be found in my material in more than 50% of all cases proved by operation". Akerlund's high figures may be partly attributed to the fact that he repeatedly examined cases exhibiting deformity of the duodenal cap but no ulcer, selecting times when their symptoms were most severe, and he comments that "at one time or another" these patients were seen to have ulcer craters. That is to say—it is likely that at many of the examinations no ulcer was found although ulceration was present.

(2) *On How Crater Size Varies with Symptoms*

After the crater has been shown, all workers agree that in general its size varies in step with the severity of the symptoms. Almost all of the same authors point out that there are exceptions to this rule, that the crater may remain or even enlarge, although symptoms vanish, and that symptoms may remain after the crater has disappeared. Akerlund says: "We can . . . follow the successive reduction of an ulcer crater and its final disappearance under a successful ulcer treatment; we can see other cases where the ulcer crater has defied all

internal therapy; we can, by repeated control examinations, follow cases for long periods and see the one ulcer crater succeed the other after a longer or shorter symptomless interval." In another paper (1931) he comments that the symptom-change is not always reflected in the crater size. Berg (1930) says: "One is occasionally taken aback during control of therapeutic results (as emphasized by Walko) when the remission in clinical findings and the X-ray appearances do not run parallel. There is no doubt that pain often disappears while the niche is still visible. At other times the niche is invisible and in spite of this the pain is present."

The frequency with which the crater alters in step with the symptoms is also attested by Shattuck (1921), Crohn and his associates (1925) and Geyman (1932).

(3) *On the Variation of Duodenal Deformity with the Symptoms*

Emery and Monroe (1931) studied 52 cases with duodenal deformity, for periods ranging from a few months to several years, and were unable to find any radiological sign upon which to assess healing. The deformity changed in 13 instances and there was doubt in 2. In 32 there was no change. The radiological and clinical alterations did not run parallel. Templeton, Marcovich and Heinz (1938) have commented more fully than most authors on this subject. In their series of 963 cases with a past or present history of duodenal ulceration, there were as far as we can calculate 314 patients with peptic ulcer symptoms, showing only deformity. How many of these were followed up we do not know, but on the evidence of those that were, and on observation of the deformity where craters were also present, they found "During healing no appreciable change in the bulb deformity . . . in the majority . . .". "When the degree of deformity did change, decrease was more frequent than increase." Geyman could not distinguish the deformity before treatment from that when the patient was symptom-free.

(4) *On the Indirect Signs of Ulceration and of Healing*

Older authors, not having their attention concentrated to the same extent upon the demonstration of an ulcer niche were more concerned in assessing activity by observation of hypermotility, pylorospasm and the quantity of resting fluid.

Friedenwald and Baetjer, writing in 1913, considered that they could determine healing of a duodenal ulcer by the loss of previously noted hypermotility. White (1919) relied on changes in gastric motility to indicate ulcer healing. Shattuck (1921) in following the progress of 7 cases of duodenal ulceration through one or two years showed "marked improvement in the filling defect" (in the duodenal bulb) "and a diminution of abnormal peristalsis with a return towards normality of gastric emptying-time" in five. Karl Walko (1926) was of the opposite opinion. A translation of his comment is that "The secretory and motor behaviour of the stomach as shown by the indirect radiological signs of ulcer alter little or not at all during the painful and latent phases in duodenal and gastric ulcer". His observations are based on a study of 155 cases; but since he failed to distinguish in his findings between gastric and duodenal ulcer his work is not conclusive.

Geyman, in 1932, having discussed the value of demonstrating the ulcer niche, comments : "Roentgenoscopically there may be a change in the picture; a pylorospasm may have disappeared or a cap irritability originally noted may not be in evidence; but the film evidence will be much the same."

(5) *On the Usefulness of Radiological Attempts to Demonstrate Healing of a Duodenal Ulcer*

We have been unable to find any critical radiological work on the usefulness of radiological attempts to demonstrate healing of a duodenal ulcer. However, many of the authors we have quoted have *stated* that re-examination is valuable, and it is widely assumed that there is something to be gained by confirming the therapeutic results with observation of the barium-filled duodenal cap. This belief is more firmly held when an ulcer crater has been demonstrated. The authors who have noted the unchanging nature of duodenal deformity have tacitly thrown doubt upon the value of radiologically controlling the treatment of duodenal ulcer when the crater itself was not demonstrated.

The physician's approach to the subject has been summarized as follows (Price's Textbook of Medicine). Speaking of both gastric and duodenal ulcers, the authors (Sir Arthur Hurst: Dr. Thomas Hunt) say: "X-ray control of healing is advisable and radiographs at four and six weeks are ideal, but not essential, if they can be obtained. There is no fixed rule which can decide exactly when an ulcer has fully healed, as the crater may become filled with granulation tissue so that no niche is seen with the X-rays for a fortnight or more before it is replaced by mucous membrane." They note that re-examination by X-rays need not be carried out until (*a*) there is no spontaneous pain; (*b*) no tenderness or rigidity, and (*c*) no occult blood in three consecutive stools.

Dr. Avery Jones (Personal communication, 1952): says: "I agree that the information from a repeat X-ray is limited, but nevertheless there are indications from the clinical angle which may not at once be apparent to the radiologist. The intelligent patient, who has had a protracted course of bed-rest and diet, expects to know what has happened to his ulcer. If the symptoms persist, a further X-ray may be necessary to persuade the patient that an operation is really necessary. It may also be necessary to persuade the surgeon to do the operation. If the symptoms persist, particularly if there are slightly unusual features, it is a good routine to review the case afresh both from the clinical and radiological angle. One cannot assume that symptoms have necessarily been related to a duodenal deformity—and hiatus hernia, gastric ulcer, pyloric neoplasm, carcinoma of the pancreas or cæcum may be discovered on reinvestigation. I am always a little suspicious of patients who have had reputed duodenal ulcers long ago and who turn up with fresh symptoms."

Summary of Information from Literature

(1) It should be possible for an average careful worker to find the actual ulcer crater in about 50% of patients with clinical and radiological evidence of duodenal ulceration.

(2) If the crater is found, it is usually but not always seen to get smaller under treatment; and it

may persist after the patient is symptom free. How often this happens and how close is the parallelism is unstated.

(3) Changes in the duodenal cap deformity during or following treatment are slight.

(4) The indirect signs were thought at one time to provide good evidence of activity; but tend now to be ignored.

Personal Observations

Material

This investigation was carried out on consecutive patients with symptoms consistent with duodenal ulceration at the time of the first examination. The great majority of patients complained of epigastric pain before meals and eased by food. Some of them had had melæna and/or hæmatemesis. A very few had had painless bleeding.

15 patients who showed radiological evidence of duodenal ulceration, but whose symptoms did not include any of those just mentioned, were excluded. Their symptoms were: (*a*) Severe continuous pain. (*b*) Indefinite dyspepsia, not related to meals. (*c*) Flatulence. (*d*) Anorexia.

5 patients with normal findings were also rejected.

About 5 others were eliminated because satisfactory radiographs were not obtained owing to obesity or excess of resting fluid or, occasionally, because we could not determine the relation of the pylorus to a visible ulcer crater.

There remained 134 patients who showed radiological evidence of duodenal ulceration; and they were divided into two groups. The first group numbering 63 patients, exhibited definite duodenal ulcer craters. The second group of 71 patients showed duodenal cap deformity only, but in them no ulcer crater could be identified with certainty.

Technique of Examination

The screen examinations were done by one or other of us acting alone. The technique of the actual examination of the duodenal cap was that of aimed dose compression with serial radiography, as described by Akerlund, Berg and others.

Re-examination

Following Treatment

Re-examination was carried out by either one of us as convenient and we frequently re-examined each other's cases. The patients came back either at the clinician's request or after we had written for them. No attempt was made at selection.

When duodenal deformity only had been discovered, we attempted to take our pictures at angles comparable with those of the previous examination.

In patients in whom the duodenal ulcer crater was demonstrated on the films, re-examinations were made at intervals varying between six weeks and two years. Rather more than half were re-examined after intervals varying between two and six months, and the numbers re-examined after two, three, four, five and six months were about equal.

Those re-examined after longer intervals fell equally into the three categories of "crater unchanged", "crater smaller" and "crater larger".

In patients in whom no ulcer crater was identified with certainty, but who had duodenal cap deformity, re-examinations were made at intervals varying between two months and three years. Half the re-examinations took place after between two and six months, and the numbers re-examined after two, three, four, five and six months were about equal. All patients, except 1, re-examined after a longer interval than six months showed no radiological change.

Inspection of X-ray Films

If a patient shows duodenal deformity and has symptoms sufficiently severe to warrant operation, the probability that he has an active ulcer is extremely high. But it is only too easy to label as a crater a small persistent fleck of barium lying in a rugal fold. At operation it is often impossible to identify the crater with any particular barium shadow. In some of the published work it is doubtful that such identification has always been attempted; and it is probable that in a number of cases the true crater has gone unrecognized by radiological examination, though the result has been recorded as one in which surgery has confirmed the radiologist's findings.

We were interested in the number of craters that we could recognize with fair confidence. If there was doubt as to the crater we felt that progress could not be reliably estimated from changes in size of the doubtful shadow. The films were inspected by both observers at the same session. It was the task of each of us to act as Devil's Advocate and throw doubt upon the findings of the other. The arguments were vigorous. If the two observers did not agree that the criteria for demonstration of an ulcer crater had been stringently adhered to, the case was placed in the "No ulcer seen with certainty" category; and if even the deformities were not perfectly convincing, the case was rejected.

The 134 cases in this investigation are those which survived the dispute.

Criteria for Diagnosis

(1) *Ulcer crater.*—The criteria upon which we based our recognition of an ulcer crater were those now universally adopted. They were described in detail by Akerlund, Cole, Berg, Haudek and Simon among others.

In particular we required one or more of the following appearances:

(*a*) A barium fleck with a translucent ring indicating a definite surround of œdema, and frequently with convergence of rugæ or other rugal deformity.

(*b*) A large residual deposit of barium which was persistent in the compression views.

(*c*) A niche projection with or without compression which was persistent in both size and shape and protruded beyond the contour of the cap.

(2) "*Cap deformity only*".—The description "cap deformity only" applies to those cases showing constant deformity of one of the types described by Akerlund. The normal rugal folds of the duodenum during peristalsis are sometimes confusing and for this reason it was necessary to satisfy the Devil's Advocate with a picture of the duodenal bulb distended to its full capacity with barium, and it was also necessary to demonstrate that the shape of the bulb retained the abnormality throughout the series of views.

Provision was made in the chart devised for recording results for coarse or deformed rugæ without an incisura in the contour of the cap; but so few of these cases were convincing after argument that they have not merited separate mention.

The Frequency of Demonstration of a Duodenal Ulcer Crater

63 of our 134 patients exhibited duodenal ulcer craters according to the above criteria (47%). The ulcer crater was only seen with certainty at a second or subsequent examination in six of these 63 patients.

17 of our patients at the time of writing have come to operation (Table I). All of these were shown to have ulcerated caps. However, the ulcer could only be examined by the pathologist in 12 because

Table I.—Findings at Gastrectomy

	Ulcer crater seen on X-ray	No crater seen with certainty on X-ray. Deformed duodenal cap only
No accurate pathology possible, but duodenal ulcer seen by surgeon	1	4
Ulcer crater examined in the resected specimen	9	3
Total	10	7

the surgeon not infrequently was forced to place his clamp across the ulcerated part of the duodenum.

Of the twelve ulcers of the duodenum which were clearly shown in the resected specimens, three were found in patients in whom we had seen only duodenal deformity, and had been unable to identify any shadow with certainty as a crater.

Since we were not particularly concerned with the proof of the diagnosis of ulceration, we took no special steps to correlate the appearances of the pathological specimen and the barium shadows. But some of the reasons for failure to recognize duodenal ulcer craters on radiological examination may be re-stated.

Sometimes a depression lined with mucosa may remain at the site of a crater. There may be a small erosion in its depth. Upon radiological examination it may appear as a large ulcer; while the pathologist, speaking correctly of a break in continuity of the surface, reports only a shallow ulcer crater no deeper than the submucosa.

At other times a wide area of a pouch-like fornix in a deformed cap may have no remaining healthy mucous membrane. The pathologist sees an ulcer more than a centimetre in diameter; but the radiologist finds it difficult or impossible to distinguish the crater. All he can be certain of is the great deformity.

Pathological estimates of the depth of an ulcer in millimetres are probably unreliable because of the disappearance of œdema from the margin in the dead specimen; but a knowledge of the ulcer depth is necessary for the radiologist who wishes to check on his findings. In an undeformed duodenal cap a shallow erosion may show at the radiological examination with compression technique; but in a grossly deformed duodenum there are so many irregular folds that one may not be justified in picking one shadow from another and calling it an ulcer.

How Crater Size Varies with Symptoms

In our series, of the 63 patients with ulcer craters demonstrated, there were 38 whose progress we were able to assess by repeated barium meals. The changes which we noted are shown in Table II,

TABLE II.—ULCER CRATER SEEN ON X-RAY

Record of Progress

No. of patients re-examined = 38.			No. of re-examinations = 42.	
	Crater larger	Crater unchanged	Crater smaller but present
Symptoms worse	4	2	—
Symptoms unchanged	2	5	1
Symptoms less or gone	1	6	21

Underlined figures indicate changes in symptoms and X-ray appearances running parallel.

which records each separate observation. At 30 of these re-examinations there was a parallel change in the size of the crater and the severity of the pain. Analysis of the remaining 12 re-examinations (which provided results not paralleled by clinical findings) is needed.

The group of 12 patients may be divided in two. Firstly there are those 5 whose symptoms remained the same, or increased. It was clinically apparent that their ulcers were unhealed, and that they were resistant to medical treatment. This was also proved directly at gastrectomy in 4. The radiological re-examination confirmed the failure to improve in 3, and was perhaps misleading in the fourth.

Secondly there are 6 patients whose symptoms diminished while their craters, as demonstrated by barium meal, remained the same. None of these had entirely lost his pain. This group illustrates the time-lag which is not uncommonly seen between clinical and radiological improvement. It should be noted that all these patients were known to have ulceration still unhealed, because of the persistence of pain. A later manifestation of the time-lag is the persistence of a crater after all symptoms have gone. Such was the twelfth patient. His pain had vanished, yet we found his ulcer bigger. He was subjected to partial gastrectomy; but shortly before his operation there was some return of symptoms, with vomiting. At operation the ulcer crater was clearly seen.

Where the clinical and radiological changes are not in step, either symptoms persist and one is forced to the conclusion that the ulcer is unhealed regardless of its radiological appearance, or there is radiological evidence of a time-lag in the healing after symptoms have diminished and gone.

CAP DEFORMITY ONLY

On 77 patients placed at first in this group we were able to re-examine 50, a number of them more than once, and at intervals ranging from one month to three years. As previously mentioned 6 of these patients were found to show ulcer craters at the second or subsequent examinations. Table III

TABLE III.—RECORD OF CASES IN WHICH NO CRATER WAS SEEN WITH CERTAINTY

No. of patients = 50.			No. of re-examinations = 55.	
	Duodenal deformity worse	Duodenal deformity unchanged	Duodenal deformity less
Symptoms worse	—	1	—
Symptoms unchanged	—	16	—
Symptoms less or gone	2	35	1

records the observations on the duodenal cap deformity. The duodenal deformity altered little or not at all while symptoms improved or remained stationary. 14 patients completely lost their pain, but retained an unchanged duodenal deformity. If the pain had fluctuated between examinations, but when we saw the patient for a second time it was of the same degree as at the first examination, it was recorded as "unchanged". On only 3 occasions did we discover any radiological change; in 2 cases the deformity having increased although the symptoms had lessened. In the other, both the symptoms and the deformity had diminished.

INDIRECT SIGNS OF ACTIVITY

The indirect signs of ulceration—hypermotility, initial spasm of the pylorus and more than a trace of resting fluid were recorded, but were so rarely present in a gross degree, and scattered in such a random fashion throughout all groups, as to be of no assistance in determining the activity or the presence of an ulcer.

THE USEFULNESS OF RE-EXAMINATION IN PATIENTS WITH A DEMONSTRATED ULCER CRATER

(1) *While symptoms are present.*—Since the radiological method of demonstrating duodenal ulcer craters is far from foolproof, failure to show a crater in the presence of symptoms cannot be taken as an indication of healing.

The radiologist can help the clinician very little unless the original diagnosis is in doubt. It may be thought of prognostic value to show whether the crater is getting smaller; but such a demonstration has no bearing on the length of time for which the crater remains visible.

(2) *After clinical cure or remission.*—Only about 50% of duodenal ulcer craters are discovered by the barium meal; duodenal ulcers are not uncommonly multiple (Berg, 1925), and in an unknown number of patients ulcers may remain still radiologically invisible in scarred duodenal caps after all signs and symptoms are gone. If, from observation of duodenal deformity and indirect signs, we are unable to demonstrate healing, the chance of re-examination being useful is reduced. The reduction is in proportion to the number of second and undiscovered ulcers. We cannot be sure that the unseen ulcer will heal at the same rate as the observed one. Moreover, that the disappearance of a niche indicates complete healing of the crater has never been proved; in fact it is probable that the lesion persists as a shallow erosion after the crater has become invisible on the pictures.

However, if we adhere to the quoted medical indication for a fresh radiological examination, that is to say, when symptoms and signs have disappeared, the question of the reliability and usefulness of the X-ray examination must be considered from a different angle.

What are the radiological signs when the patient is clinically cured, and is there ever still evidence of activity?

Among our patients whose symptoms improved under treatment while their craters grew smaller, there are 7 who were re-examined after all symptoms had disappeared. The times of these examinations after the first barium meals, were six weeks, two months, two months, three months, four months and six months respectively, and in each case the crater, though smaller, persisted. To these 7 must be added one previously mentioned whose symptoms vanished although the ulcer became larger.

Therefore, in a very high proportion there is radiological evidence of an active ulcer after all symptoms have gone. Even so, there can only be an indication for radiological re-examination when the clinician's actions can be influenced by the radiological finding of the persistence of the crater.

If, for instance, he is prepared to readmit the patient who is symptom free; or to advise surgery or a continuation of a strict regime, which would otherwise be relaxed, there is a place for re-examination of the apparently cured, that is to say, the patient whose symptoms have yielded completely to treatment.

But we do not know whether the continuation of strict treatment until the ulcer is radiologically invisible is both clinically worth while for the prevention of relapses, and economically possible for the patient confined to bed and the hospital that keeps him. It is not unlikely that the patient would need to be treated rigorously for many extra weeks.

We have not even the radiological data to show how long in the average case strict treatment is necessary before the crater vanishes. In our experience it is a great rarity to follow an ulcer to complete healing, and we do not really know whether this moment of healing marks any stage in the disease allowing an alteration of treatment.

Much of our material has been drawn from out-patients, but it is among the in-patients confined to bed in hospital that most benefit from radiological re-examination might be expected; for a large proportion of them become symptom-free after a very short time.

To sum up this section it may be said that we do not know whether it is ever useful to re-X-ray a patient when his symptoms have gone. But certainly unless the clinician is going to act upon our demonstration of a persistent crater in the symptom-free, radiological re-examination is a waste of time.

(3) *After relapse.*—If the diagnosis of duodenal ulceration with a visible crater has been established by a radiological examination at a previous period of pain, and satisfactory X-ray films are available, and the symptoms are exactly the same, there is no purely diagnostic value in a re-examination when the pain begins again.

The indications for radiological re-examination, after the relapse has apparently yielded to treatment, are the same as after the first attack.

The Value of Re-examination Where Cap Deformity Only was Discovered

If there is a deformity present but no crater seen with certainty, further radiological examination does not provide the clinician with any estimate of healing. A subsequent barium meal will nearly always be valueless.

Occasionally, an ulcer crater may appear at subsequent examinations.

In this small series in only 8% of cases with duodenal cap deformity did re-examination reveal an ulcer crater (1 of our 6 cases showed an ulcer only at the fifth re-examination).

Until we know the value of persisting with strict treatment until all demonstrated craters have vanished, it is economically unsound to re-examine patients who show only cap deformity, to discover about 8 in a 100 who subsequently develop craters, in order to perform further barium meals of questionable value on the 8 when their symptoms have yielded to treatment.

Indications for X-ray Examination

The indications for radiological examinations in duodenal ulceration are, firstly, for diagnosis.

Secondly, perhaps, and at the discretion of the physician who is prepared to act upon the finding of a persistent crater, when an ulcer crater was demonstrated at the first examination and the patient has become symptom-free under treatment.

Thirdly, with the same reservations, in a relapsed patient (who has not had a barium meal at the beginning of the relapse because his diagnosis was established in the past) when he becomes symptom-free. If an ulcer crater is then visible, he may be benefited by further strict treatment.

Fourthly, when the nature of the symptoms changes.

There is *no* indication for the re-examination of patients in whom duodenal ulcer craters were demonstrated while their symptoms remain, if the diagnosis is not in doubt (and surely there are great numbers of patients in whom the diagnosis is truly in no doubt?).

There is at present *no* indication for the re-examination of patients during treatment in whom no ulcer has been seen with certainty, but in whom the clinical diagnosis was confirmed by the presence of duodenal cap deformity.

There is at present *no* indication for a diagnostic barium meal in a known sufferer from duodenal ulceration when his symptoms recur after a period of freedom from pain, and the films are available for inspection.

When it is thought that the patient requires a partial gastrectomy for recurrent or continued symptoms, and if the radiological diagnosis has once been firmly established, little store should be set by the demonstration of a crater immediately prior to operation, in a cap known to be deformed.

We cannot agree that the patient's wish alone is a valid reason for a radiological examination.

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Section of Physical Medicine

President—DONALD WILSON, M.B., M.R.C.P.

[*March* 11, 1953]

Methods of Electrodiagnosis and Electrotherapy by Stimulation

By KARL M. WALTHARD, M.D.(Geneva)

I HAVE chosen to discuss "methods of electrodiagnosis and electrotherapy by stimulation", as I feel the subject has been rather neglected of late in favour of the newer electrodiagnostic method by detection, electromyography. I have no personal experience of this newer method as it has not yet been introduced in our clinical and hospital work. We have always felt it to be of scientific value for the investigation of muscle activity rather than of clinical importance, whilst the older method of nerve and muscle examination by electrical stimulation is still of great clinical importance.

I shall describe our methods at Geneva. I always had it in mind to develop a simple technique and to have an apparatus constructed which could be performed simply and quickly by the practitioner because, in my opinion, the electrodiagnostic examination should not be in the hands only of the specialist. It is, of course, necessary to get some experience in this special field of examination, but I have noticed that my young interns are able to carry out these examinations after only a few days' instruction.

Our examinations are performed in the same way as in the past 100 years: by using two forms of electric stimulations, the old faradic current, the frequency of which varies between 1 shock and 100 shocks per second, and the direct current. To these stimulations, we have added the condenser discharges for measurement of chronaxia.

The apparatus we use at Geneva has been constructed to replace the old "Pantostat", which our predecessors used to measure the nerve and muscle excitability by determining the faradic and direct current thresholds; it seemed to me necessary to provide a modern apparatus which permits an electrodiagnostic examination based on our actual notions of nerve and muscle excitability and which is as simple as possible. Our apparatus allows all the different manipulations to be made easily and quickly. At the same time it gets the right results for clinical purposes without creating too much discomfort for the patient.

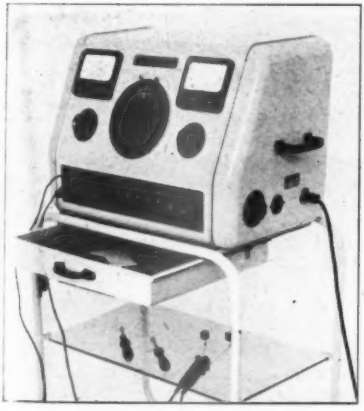

FIG. 1.

The different devices are enclosed in a metallic box 59 cm. (23·2 in.) long, 38 cm. (15 in.) wide and 38 cm. (15 in.) high. The whole apparatus weighs 24 kg. (53·5 lb.). The box is fixed on a wheeled framework and can easily be moved to the patient's bedside (Fig. 1).

On the front of the apparatus are fixed a milliammeter and voltmeter. Between them is a disc with a triple scale indicating the chronaxia in milliseconds. Under the milliammeter is the shunt 1 to 5 and 1 to 50 mA. Under the voltmeter is the switch to regulate the intensity of the faradic or direct current. At the bottom is the switchboard regulating the different currents; from right to left are the following switches (Fig. 2):

(1) Pushed up: single shocks are switched on; pushed down: stimulations are given automatically, about one per second.

(2) and (3) Pushed up: the direct current circuit is switched on for measurement of the rheobase; the values are indicated simultaneously by the milliammeter and voltmeter. Pushed down: the rheobase voltage is doubled and the chronaxia circuit switched on. Chronaxia is measured by turning the disc in the centre of the control panel of the apparatus.

(4) Pushed up: shunts in a balance resistance of 6,000 ohms; pushed down: one of 10,000 ohms in the chronaxia circuit.

(5) Pushed up: switches on the first chronaxia scale; put into 0-position: the second chronaxia scale; pushed down: the third chronaxia scale. The three scales correspond to the three circles of the disc in the centre of the control panel.

(6) Pushed up: switches on the direct current and the chronaxia circuit; pushed down: the faradic current circuit.

(7) Represents a "magic eye", small cathode ray oscillograph for control of the faradic current frequencies, 1 to 100 per second.

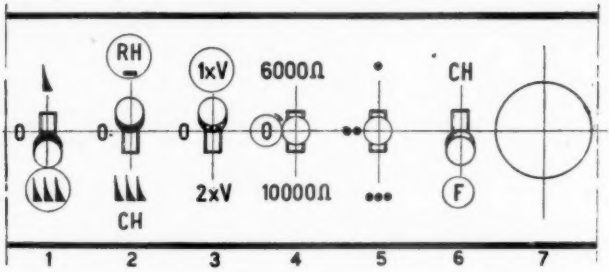

FIG. 2.

At the left of the switchboard are the contacts to fix the cables leading to the patient's electrodes. The contacts are combined with a pole-reversing switch. At the right end, the general switch is fixed. At the right side of the apparatus is the switch for regulating the faradic current frequencies, controlled by the magic eye.

The ½-in. silver electrodes are stuffed with asbestos and are supplied with a hand circuit breaker. They can be fixed quite easily on the motor point. We usually use one stimulating electrode, the indifferent large lead plate being fixed on the neck of the patient. If necessary, we use a so-called double electrode, both poles being of the same size, for longitudinal stimulation, especially when the monopolar stimulation of denervated muscles is disturbed by too strong contractions of normal muscles in the neighbourhood.

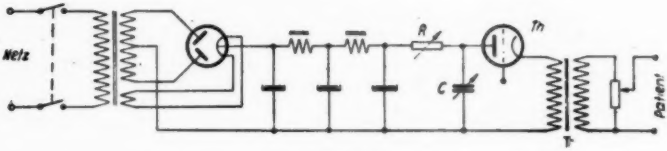

FIG. 3.

The faradic current circuit is very simple. The main feature is the replacement of the mechanical hammer interrupter by a thyratron tube interrupter and the use of condenser discharges as first coil stimulations. The pattern of the circuit (Fig. 3) shows a rectifier of the alternating net current which delivers direct (rectified) current through a rheostat (R) into variable condensers (C). The condensers discharge through the thyratron tube (Th) and the primary coil of a transformer (Tr) as soon as the necessary tension is reached to permit the passage of the current. After discharge, there is a pause until a new charge is again built up on the condenser.

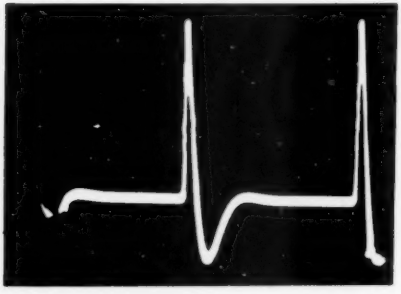

FIG. 4.

The secondary coil of the transformer is connected with the patient through a variable potentiometer.

Study by means of a cathode-ray oscillograph shows a curve which is optimal for physiological stimulation. The perpendicular rises in voltage follow each other with mathematical precision and the gradual decrease in voltage averts counter-stimulation as far as possible (Fig. 4).

The current may be altered quantitatively from slow individual impulses of 1, 4 or 9 per second to a continuous rapid vibration up to 100 per second, which can be controlled by the magic eye.

The direct current and the chronaxia circuits are illustrated in Fig. 5. There are two circuits: (1) The regular direct current circuit, with a milliammeter and a voltmeter, which is used for the measurement of the rheobase; (2) The chronaxia circuit: a condenser of a fixed capacity is charged

with the double voltage threshold which has been estimated by the measurement of the rheobase. A specially constructed interrupter closes either the circuit for charging the condenser or the condenser discharge circuit in which the patient is included.

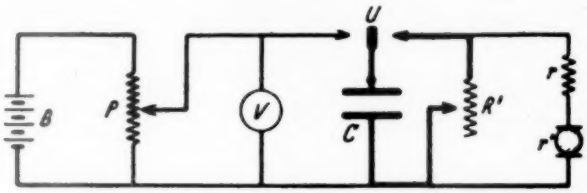

FIG. 5.

The pattern of our apparatus is different from Bourguignon's device, mainly because it was impossible to manage the original Bourguignon table with its many condensers without help. We needed an apparatus by means of which rheobase and chronaxia could be measured as easily as by the faradic examination. This goal was reached. We reduced the number of condensers to three, one with a capacity of 1 microfarad (μF.), the second with a capacity of 1 + 10 μF. and the third with a capacity of 1 + 10 + 138 μF. Nevertheless, to get the necessary variability of the discharge duration of these condensers, we introduced a variable resistance free of induction of 2,000 ohms into the discharge circuit. Parallel to this resistance R′, two other resistances r (6,000 or 10,000 ohms) plus r′, (patient's resistance) are shunted in series. The total resistance of the discharge circuits has been calculated according to the formula of the distribution rule of Kirchoff

$$R = \frac{R'(r + r')}{R' + (r + r')}$$

Using this formula, the most favourable relationship between the resistance R′ and r + r′ has been determined. Under these conditions, the measuring error does not exceed 10% by the patients' resistances which may vary between 100 and 20,000 ohms.

In practice, chronaxia is not measured directly. We measure the total resistance R of the discharge circuits. But, in order to read on the scale of the disc chronaxias expressed in milliseconds, the various values of the measured resistances have been empirically calculated in milliseconds with the aid of the chronaxia formula: T in milliseconds = 10^{-9} K R C (K being the constant time factor 0·37, R the measured resistance, C the capacity of the condenser in μF).

Table I illustrates the result of this calculation:

TABLE I

R′	Scale I C = 1 μF msec.	Scale II C = 11 μF msec.	Scale III C = 149 μF msec.
100	0·0368	0·405	5·48
500	0·1805	1·985	26·9
900	0·319	3·50	47·5
1,300	0·452	4·97	67·3
1,700	0·580	6·38	86·4
2,030	0·673	7·40	100·2

The three circles on the triple scale are therefore marked in chronaxias instead of resistances, the first circle giving the values of 0·03 to 0·5 msec., the second circle the values between 0·4 to 7 msec., and the third those between 5 to 100 msec. The condensers can be charged up to 250 volts.

The interrupter charging and discharging the condensers acts automatically at a frequency of about 1 per second. The hand interrupter is therefore no longer necessary.

The technique of measurement of both rheobase and chronaxia is, with this apparatus, very simple. The stimulating electrode (used for the faradic examination) being apposed on the motor or another point of the nerve or muscle, the direct current threshold is first determined by slowly increasing the current's intensity. Simultaneously, we observe the corresponding values in milliamperes and volts, until we get the minimal contraction, which may be a brisk twitch in normal muscle, or a sluggish vermicular contraction in denervated muscle. Without changing the position of the electrode the voltage is doubled (by changing the position of switches 2 and 3). The chronaxia can then be estimated by turning the disc until the same muscle contraction appears again. If necessary, immediately after the measurement of chronaxia, it is possible—again without changing the position of the electrode on the motor point—to switch back to the direct current circuit, to control the rheobase.

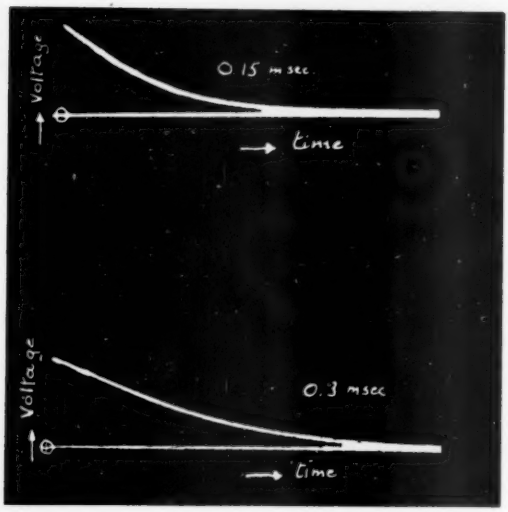

FIG. 6.

The photographic control of condenser discharges representing chronaxias taken at the same voltage but at different durations (0·15 and 0·3 msec.) by cathode-ray oscillograph is shown in Fig. 6.

Interpretation of results.—The examinations of nerves and muscles with the faradic current are interpreted merely as *qualitative* results. We want to know if a tetanic muscle contraction can be produced by application of the faradic current, by stimulation of the nerve or by the direct stimulation of the muscle. The result is *positive* or *negative*. It is not necessary to measure the intensity of the faradic current producing the tetanic muscle contraction, which in any case is difficult to determine. For the practitioner it is quite sufficient to know if a muscle paralysis is of organic or psychic origin. The single faradic spike of the faradic current we use has a duration of 1/1,000 second. This short shock represents only a very small quantity of electrical energy, just sufficient to provoke the contraction of a normal or, perhaps, a very slightly impaired muscle fibre. If the nerve or muscle damage is more serious, the muscle fibre will not respond either to this small stimulation or to a series of short shocks; the faradic excitability is abolished.

The diagnostic value of the faradic examination is shown by some personal experiences:

A soldier who was sent to my department for electrodiagnosis presented a flaccid paralysis of the left arm following a very trivial accident: he had walked into a barbed wire fence, injuring the left shoulder and sustaining several slight scratches. The military surgeon told him to be extremely careful not to infect the wounds and ordered complete rest of the arm. He rested so carefully that finally he was unable to move the arm voluntarily. The surgeon, fearing a neuritis, sent him to a military hospital and from there, after some weeks, he came to us. The application of faradic current to the left deltoid muscle proved the integrity of the muscle excitability. After the first examination he began again to move and lift the arm and after three applications of combined faradic and active muscle exercise he was completely cured.

The faradic examination can be an important guide in the differential diagnosis between neurogenic and myogenic affections.

During the war I had the medical care of about 100 soldiers suffering from tri-ortho-cresyl phosphate poisoning. Tri-ortho-cresyl phosphate is an oily substance used as machine-gun oil. It has no particular odour or taste and had been mistaken for comestible oil and used in baking cheese slices offered as supper to the company. The men who ate these dishes, after having recovered from the acute symptoms of the poisoning, developed a grave flaccid paralysis of both legs and, in several cases, also of the hands. The examination of these men showed a marked difference between the motor and the sensory disturbances: very marked paralysis, but little or no sensory disturbances. As the syndrome of that poisoning is known in the literature as a "neuritis", I was interested in the result of the electrodiagnostic examination. The result was somewhat peculiar: the faradic excitability of the lateral popliteal *nerve* was preserved, but it was not possible to get a tetanic contraction by direct muscle stimulation of the tibialis anticus or of one of the other extensors. In a neuritis, there is a contrary result: first, the faradic excitability of the nerve is abolished and only afterwards does the faradic muscle excitability disappear.

The deduction which I drew from this result was that the so-called "neuritis" is in fact not a neuritis, but a paralysis of myogenic origin; it cannot be due to a *total*, but probably to a *partial* destruction of the muscle, since the faradic stimulation of the lateral popliteal nerve still provokes tetanic contraction and muscle fibres of normal constitution must remain in normal connexion with the nerve.

Biopsy showed a very marked primary degeneration of muscle fibres, but normal muscle fibres were lying between degenerated fibres. Special silver stainings showed also that the terminal nerve plates were preserved.

I mention these cases only to illustrate that the simple faradic examination is a great help in discerning obscure motor disturbances.

A few years ago a colleague sent me an elderly lady suffering from a syndrome which resembled a lateral amyotrophic sclerosis, with double Babinski, but rather flaccid paralysed legs. The faradic examination of the leg muscles was completely normal. All muscles responded to the stimulation with a normal tetanic contraction, by indirect nerve stimulation as well as by direct muscle stimulation. I told my colleague that in my opinion the patient was suffering from a myogenic and not from a neurogenic affection. The patient died recently, and autopsy revealed Besnier-Boeck disease, which was widespread in the muscles. The nervous system, including the spinal cord, was found intact with no signs of pathological change or degeneration.

These three examples prove that faradic examinations of nerve and muscle are of great clinical value and should be used more frequently.

For clinical purposes, therefore, I believe it is unnecessary to measure rheobase and chronaxia in every case with preserved faradic nerve and muscle excitability. We usually proceed to this form of examination only when the faradic excitability is abolished.

I do not need to emphasize here the merely relative value of measurement of rheobase. The direct current threshold is not an accurate measure of nerve or muscle excitability. On the contrary, chronaxia—introduced in electrophysiology by Lapicque (1926) and adapted for clinical use by Bourguignon—is regarded as the only exact quantitative measure of it.

Interpretation of chronaxia measured with our apparatus in regard to the nerve and muscle excitability.—We first examined carefully the normal nerves and muscles, with results very similar to those of Bourguignon (1923). We observed that the normal muscle excitability measured by stimulation of the motor point is characterized by very low values, but that normal chronaxias may vary in a small range. Chronaxias between 0·05 and 0·8 msec. may be considered as practically normal; with these values the faradic excitability is preserved. Proximal and anterior muscles as a rule are characterized by lower chronaxias than distal and posterior muscles. The rheobase values vary in normal muscles between 20 and 50 volts; the nerve rheobase on the contrary may be only 10 to 20 volts. Muscle response is brisk.

Chronaxias between 0·8 and 7 msec. must be interpreted as signs of a slight nerve or muscle dysfunction caused by an organic lesion which is not very severe and—depending on the clinical course of the affection—may be harmless and of good prognosis. The faradic excitability in these cases may be diminished or abolished. The muscle contraction may be somewhat prolonged, especially the relaxation. Chronaxias of 8 msec. and over are signs of severe damage. The contraction is sluggish and vermicular; the faradic excitability is abolished.

In other words, chronaxias in the range of the first circle of the disc are to be considered as normal, those of the second as slightly pathological and those of the third circle as pathological.

The question is, are the chronaxias we obtain by nerve and muscle stimulation with condenser discharges sufficiently precise values to permit an exact appreciation of nerve and muscle condition in view of the clinical diagnosis?

To answer this question it is necessary to consider the importance of electrodiagnostic examination for clinical purposes and how far this form of examination may help to clear the diagnosis.

We all agree that the so-called classic electrodiagnostic methods, with their false quantitative faradic and direct current threshold values are obsolete and, with these methods, the term "reaction of degeneration". This expression should be abandoned entirely, as Humbert (1949) postulated at the 6th Congress of the French-speaking Electro-Radiologists. The term "reaction of degeneration" implies an interpretation: the observed fact, the sluggish muscle contraction, is interpreted as the symptom of a causative factor. In accordance with Bourguignon, Humbert therefore proposed to abandon this expression and to replace it by the term "reaction of retardation"—in French "réaction de ralentissement". The term "reaction of retardation" means nothing beyond what we observe, the slackening and sluggishness of the muscle contraction characterized by high chronaxia.

As Humbert says we have to be very careful in the interpretation of what we observe by electrical nerve and muscle stimulation. We can only describe what we see. The result of our observations has then to be compared with the result of other clinical observations. Only in this way can we come to the right appreciation of the nerve and muscle conditions we have to examine. We have to be more modest in the interpretation of our electrodiagnostic results.

Conclusion

The aim of the electrodiagnostic examination is to get a precise picture of nerve and muscle excitability from a clinical point of view. This involves three desiderata: (1) to get diagnostic value as exact as possible, (2) to get these results quickly, and (3) to cause a minimum of pain and discomfort to the patient.

These desiderata seem to me fulfilled by the combined faradic and chronaxia apparatus I have described. We get exact qualitative and quantitative results by a simple technique; the examination can be done by a single examiner without help. The stimulations used, especially in the measurement of rheobase, are as painless as possible; the condenser discharges in the measurement of chronaxia are much less painful than the stimulations by rectangular direct current impulses.

In my opinion measurement of chronaxia is sufficiently exact for clinical use, and I do not therefore use the procedure Dr. P. Bauwens has established of plotting the intensity-duration curve. I have two objections to this method: (1) As Dr. Bauwens himself mentions, the application of rectangular currents is very painful.

I tried, several years ago, with the aid of Professor Oscar Wyss (Zurich) to use a rectangular current stimulator. I tried it first on myself, but an impulse of only a few milliamperes caused such pain that I could not apply these currents to our patients. Why are rectangular stimuli so much more painful than the condenser discharges? It may be that the sensitive receptors in the skin depending on the very quickly reacting cerebrospinal nervous system react much more easily to rectangular impulses than to condenser discharges of longer duration. It may be that this is the reason why

chronaxia impulses with their exponential curve are much better tolerated and cause practically no pain. Patients examined with condenser discharges compare them with light hammer shocks, but the rectangular current impulse creates pains like a needle stitch. We therefore often begin, in patients with grave paralysis but preserved sensitivity, to try to get chronaxia first by varying the voltage and to control the rheobase afterwards.

(2) The second objection is in the nature of a question: Why plot a curve instead of getting one straight value, chronaxia? I think that it is possible to get the same results by measurement of chronaxia as by plotting the curve, and that the same conclusions may be drawn as to nerve and muscle conditions by both methods. Normal, slightly affected and gravely denervated muscles may be distinguished and characterized by different chronaxias.

I should like to ask Dr. Bauwens about the interpretation of the curve he published as Fig. 16 (p. 71) in his contribution on electro-diagnosis in Dr. F. J. Bach's book "Recent Advances in Physical Medicine" (London, 1950). The curve, according to Dr. Bauwens' interpretation "probably results from the integration of two distinct curves, one representing the aggregate response of normal fibres, the other that of denervated fibre component". But, if we draw one straight line from the point where the I/T-curve of the denervated component of the muscle leaves the basic line at the intensity threshold between 30 and 100 msec. to the top point of the I/T-curve of its normal component (the point indicating the duration at an intensity of three times the rheobase), the point situated on this straight line at an intensity of 2·5 times the intensity threshold (which corresponds to the double voltage threshold) indicates the chronaxia with a duration of about 1 msec. But, this value, for the abductor digiti minimi, is too great to be considered as normal; I would in measuring this chronaxia come to the same conclusion as Dr. Bauwens; in this case there exists only a slight disturbance of muscle excitability.

I do not wish to criticize Dr. Bauwens' method of plotting intensity-duration curves. He is accustomed to his method, which is excellent and gives him entire satisfaction. I only want to point out that in modern electrodiagnostic examinations different methods may be used giving similar results.

Electrotherapy.—The apparatus described may also be used for treatment. We may apply the faradic impulses as well as the condenser discharges for practising medico-electrical gymnastics and muscle stimulation. The automatic interrupter will provide regular stimuli at the rate of about 1 per second. This rhythm would not too quickly fatigue the muscle. I have not found that we gain more by the application of a so-called undulating current than by the simple brisk interrupter. What we need is an adequate faradic or condenser discharge output producing a vigorous muscle contraction. When the faradic current is used, we apply a stimulus somewhat higher than the threshold intensity in order to produce a fair tetanic contraction. To train denervated muscles we use condenser discharges of a greater duration than chronaxia; in other cases we may increase the voltage.

For these treatments we use quadrangular electrodes which can be fixed on the muscles with rubber bands.

I have explained how we in Geneva get an electrodiagnosis for clinical purposes. I realize that we cannot work like the physiologist. In my opinion we, as clinicians, have to compromise between the utmost exactitude of the scientist and care for the patient. I hope I have found that compromise in the apparatus I have described.

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Dr. P. Bauwens, in reply to Professor Walthard, said that with regard to the plotting of intensity duration curves, it must be agreed that these represent the full facts concerning the excitability characteristic of muscle and nerve while such measurements of chronaxia or of ratio of the galvanic and faradic thresholds are merely abbreviations which cannot claim to show the profile of a curve though they may hint at it.

As to the greater discomfort caused by the current-stabilized rectangular waves, Professor Walthard was kinder than some of his critics who had found this method impossible to use. He consoled himself with the idea that for a matter of fifteen years he had been achieving the impossible.

Dr. A. T. Richardson: While I agree with Professor Walthard that the study of the responses of nerve and muscle to electrical stimulation is of great clinical importance, I would emphasize that such an electrodiagnostic procedure can only supply evidence of denervation in total or in part. Electromyography, however, in combination with "stimulation methods", can supply evidence of the location of a lesion within the motor unit as well as early evidence of regeneration of lower motor neurones. We have found at St. Thomas's Hospital that the combined investigation is of particular value in those cases in which muscle biopsies fail to exhibit any specific histological changes.

Professor Walthard's finding of a myogenic lesion in cases of tri-ortho-cresyl phosphate poisoning is of particular interest in view of the recent occurrences in this country of poisoning from a new organic phosphorous insecticide, mipafox. The clinical syndrome produced by this insecticide closely resembles that produced by tri-ortho-cresyl phosphate. However, the electrodiagnostic picture is that of a polyneuritis. One of the recent cases described by Bidstrup, Bonnell and Beckett (1953, in press) also exhibited a profound disturbance of neuromuscular transmission in the early stages, possibly related to the inhibition of cholinesterase by this substance.

Section of Psychiatry

President—The Hon. W. S. MACLAY, O.B.E., M.D., F.R.C.P., D.T.M. & H., D.P.M.

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Anorexia Nervosa: A Study in Prognosis

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MANY of the clinical features of anorexia nervosa are well known, but its natural history, that is the course of the disorder over a number of years and the eventual outcome, has been less adequately studied. Those accounts which are available are to some extent conflicting (Venables, 1930; Ross, 1936; Nemiah, 1950). At the suggestion of Dr. Denis Leigh, an attempt was made to trace patients who had previously been admitted to hospital with this disorder. We were concerned to assess their present psychiatric status, and to evaluate, if possible, the effects of the various treatments used, particularly psychotherapy. During the investigation, we have necessarily formed some opinion as to the nature of anorexia nervosa, and its place in psychiatry.

MATERIAL AND METHOD OF SELECTION

38 patients provide the case material. 37 of these had been admitted to the Maudsley Hospital during the last twenty years, and one was seen elsewhere. 30 patients left hospital with a diagnosis of anorexia nervosa. The remainder were diagnosed as either endocrine disorder, or neuroses. In retrospect it appears that these 8 patients could, in fact, have been regarded as suffering from anorexia nervosa, and that the original diagnosis was either mistaken, or that more general psychiatric terms were preferred.

Sex.—34 patients were women and 4 men. In the literature, males form from 5 to 30% of all sizeable groups, except when excluded by the author's own criteria. There are no adequate grounds in the present state of knowledge for arbitrarily omitting them.

Marital state.—At the time of admission 6 patients were married.

Age-distribution.—In 70% of the patients the onset of the illness was before the age of 26, most frequently during the years from 16 to 20. 5 patients were aged 15 or less, and 3 were over the age of 35.

Duration of illness.—At the time of admission to hospital, half the patients had been ill for two years or longer, and about 20% for five years or more. In fact, half had received treatment in other hospitals. Our material, then, was to some extent already selected for chronicity of illness. But as anorexia nervosa tends to be a chronic malady, a duration of five years or over is not unusual.

THE SYMPTOMATOLOGY

Before discussing further the general psychiatric picture, some points of special interest may now be considered. In the first place, there are important theoretical implications to be drawn from the *temporal relationship of the three main symptoms.* Amenorrhœa which arises as a result of psychological disturbance is held to be due to failure of the anterior pituitary to secrete a trophic sex hormone. One link in the chain is the hypothalamus; in fact the term "hypothalamic amenorrhœa" has been used to describe this kind of menstrual disorder. The question arises, does this alleged deficiency of the pituitary cause, or contribute to, the anorexia and loss of weight, as well as the amenorrhœa? This is a hypothesis which has attracted several authors (Sheldon, 1937; Decourt and Michard, 1949). If the answer is in the affirmative, anorexia nervosa is a clear-cut psychosomatic entity.

It seems to us that the clinical facts are against such a possibility. Considering first the anorexia and the amenorrhœa, we find that, in our material, the amenorrhœa preceded the anorexia or was contemporaneous with it in half the cases; in the remainder, amenorrhœa followed anorexia, by months or even years. Now amenorrhœa is said to be the most sensitive indicator of hypopituitarism (Hubble, 1952); so it is likely that, at any rate in half the cases, the anorexia has a different origin.

The time of onset of the weight loss is harder to place. Some French workers have held that loss of weight, together with amenorrhœa, may precede the anorexia, and therefore have an

endocrine origin. Judging from what we have learnt of the habits of our patients, it seems more likely that the French anorexics were capable of deceiving their relatives, and their physicians, as to their actual food intake. In our experience, restriction of diet always preceded the loss of weight. It is worth pointing out here that marked loss of weight is by no means a constant feature of pituitary deficiency, in which weight often increases. The shades of Simmonds' cachexia seem still to linger round this subject.

Moreover, a *study of the anorexia* reveals that it is not simply a failure of appetite; it is on the contrary, a positive symptom, an active repugnance for food, associated with morbid ideas about the effects of eating or not eating. Such ideas and the behaviour to which they give rise are familiar and need not be recounted here. But they tend to persist even after years. A spinster of 45 had been anorexic since the age of 11. During interview when the subject of food was discussed, she flushed, shrank away and asked for a change of topic. It was as if the conversation had become indelicate. To the best of our knowledge, such peculiarities of behaviour are not found in organic pituitary disease.

Apart from the question of functional pituitary deficiency, it has been suggested that the amenorrhœa in these patients is related to constitutional endocrine factors, such as hypogonadism. We therefore studied the time of the *menarche* in our material. Only 5 out of 30 women in whom information was available, began to menstruate after the age of 15. Moreover, in the great majority of cases, menstruation was established normally before the onset of the illness. Clinical facts such as these suggest to us that anorexia nervosa is not a specific psychosomatic syndrome. On the other hand, we believe that the triad of symptoms, anorexia, amenorrhœa and loss of weight occurs in several different psychiatric settings. The common factor is the presence of morbid ideas about food.

Personality traits.—The psychiatric setting may be illustrated first by the personality traits of this group of patients. In Fig. 1 some of these are shown. About half the patients have *obsessional traits.* A tendency to excessive interest in bowel and eating functions is shown under the heading *alimentary preoccupations.* Now these patients have been held to possess a special kind of personality, the "anorexic personality," similar to the anal-erotic character of Freud (DuBois, 1949). But, in fact, only about half those with alimentary preoccupations described obsessional traits; and many with the latter did not show unusual interest in food or in their bowels. So, after all, we are afforded only a glimpse of this special anorexic personality.

Similarly, other traits described as characteristic of these patients, such as *conscientiousness, ambition and exceptional energy* (Rahman *et al.*, 1939) tend to wane in significance when the group as a whole is examined. These traits are here found only in a minority, and are balanced to some degree by their opposites.

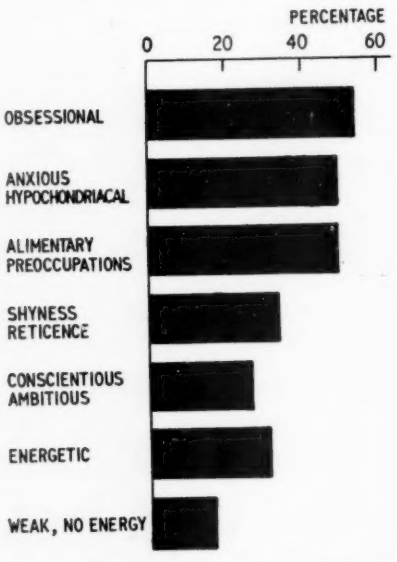

FIG. 1.—Incidence of various Personality Traits in 38 cases of anorexia nervosa.

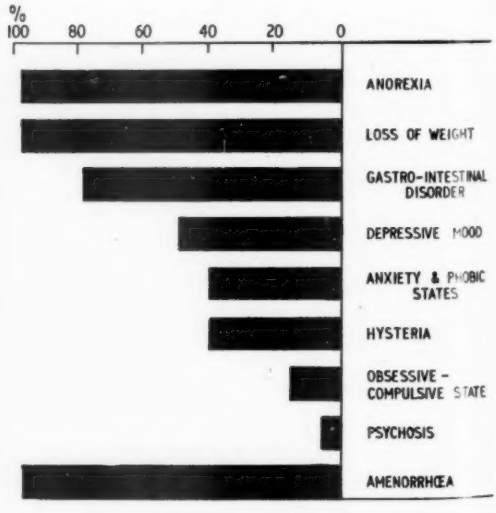

FIG. 2.—Incidence of various symptoms in 38 cases of anorexia.

The traits grouped together under the heading *anxious, hypochondriacal,* reflect the disposition of many of the group to develop neurotic symptoms. In fact, 3 out of 5 showed neurotic traits in childhood; and half the patients experienced neurotic illness later in life, but before the onset of anorexia nervosa.

It appears, then, that no common personality type can be defined. We can only say that there is an absence of integrated, normally developing personalities. The group as a whole shows personality difficulties, but the individual traits differ from patient to patient.

Psychiatric Symptomatology.—Apart from the anorexia, amenorrhœa and loss of weight, which are the definitive symptoms, there are striking differences in the symptomatology shown by individual patients (Fig. 2). These have been somewhat disregarded in the literature. Depression is common; indeed in older patients with anorexia, the condition is usually thought of as essentially a depressive illness. But some of these younger patients are depressed too. They have a persistent mood change, sometimes with feelings of hopelessness, or of unworthiness, and sometimes with suicidal preoccupation. 5 in fact made suicidal attempts. To regard all these as hysterical would be to risk underestimating their gravity.

A woman of 23, after a broken engagement, developed amenorrhœa and anorexia. She thought herself too fat, her clothes felt too tight, she was afraid to eat and vomited when food was forced on her. She attempted suicide by swallowing slimming tablets. On admission to hospital a diagnosis of anorexia nervosa was made. She was tube-fed, but vomited. She did not improve during a stay of three months. At the present time her illness has lasted without intermission for sixteen years. She is very depressed and hopeless, and weighs only 5½ st.

Here is another case of quite a different kind:

A patient aged 24 refused to eat and lost 4 st. She became amenorrhœic. There had been a similar illness at the age of 15. The diagnosis was anorexia nervosa. Further exploration revealed an obsessive-compulsive neurosis dating from childhood. This patient was later very much improved by leucotomy.

I need not labour the point that these patients do not all fall into one particular diagnostic category. The psychiatric setting varies. There is no neurosis specific to anorexia nervosa, and no specific anorexia nervosa.

Treatment and Immediate Outcome

The orthodox treatment, by *supervised diet,* was the one most often used, and was employed at some stage in 60% of the patients. *Modified insulin* was frequently used as an adjunct to dietary methods. *Endocrine* preparations of various kinds were given in 30%. Including therapy given in other hospitals, 25% (9 patients) received *E.C.T., leucotomy,* or *insulin shock.* 2 of the 3 patients who had leucotomy benefited, but the follow-up periods do not exceed three years. If, as we believe, most of these patients have personality difficulties, good long-term results following leucotomy are not to be expected. One patient gained a stone during a course of E.C.T. In another case, no improvement occurred after 15 deep insulin comas. A few had no treatment, because they either died, or else recovered, too soon. 30% received some form of *psychotherapy,* excluding simple reassurance and persuasion.

The results of psychotherapy are of interest because of the rather specific psychopathology recently put forward for anorexia nervosa, by some American psychiatrists (Waller, *et al.,* 1940). We have already remarked that we consider the symptoms anorexia, amenorrhœa and loss of weight do not form a specific syndrome based on endocrine dysfunction. Is it possible that they are linked by a common psychopathology? Briefly, according to these authors, the fear of eating results from phantasies of oral impregnation; the fear of growing fat is associated with fear of pregnancy; and constipation and amenorrhœa are also related to pregnancy phantasies. So, to these patients, eating, gaining weight and menstruation are all sexual functions. Anorexia nervosa is therefore fundamentally a sexual neurosis. Depending on one's allegiances, it could be held that psychotherapy would be the treatment of choice.

In our material, 8 patients received systematic psychotherapy, consisting of regular interviews, spread over a period of months, and directed toward giving the patient some conscious awareness of the meaning of her emotional problems. Interpretations had been given in some cases. Only one patient derived lasting benefit from this treatment. Of the others, 3 in whom insight therapy was attempted, broke it off by running away or refusing to see the therapist. These must be accounted failures of therapy. Of the remaining 4, 1 patient improved at first and was transferred to the out-patient department; but she died rapidly with a fulminating tuberculosis. The other 3 patients made no improvement; but 2 benefited later from physical methods, one from E.C.T., the other from leucotomy.

The effects of psychotherapy are notoriously difficult to assess. Moreover, in the present series, only about one-fifth did in fact receive any systematic psychotherapy. So the poor results we have observed must be seen in proper perspective. On the other hand psychotherapy was available for a larger number of patients, if adequate contact, as a basis for it, could have been established. That it could not, shows that psychotherapy, of the kind now under discussion, is often impracticable.

Ross (1936) published the only follow-up study on this particular aspect of the disorder. One of his 17 patients died, and 5 relapsed after receiving psychotherapy, but eventually 16 did well. Hurst (1936) and Venables (1930) claimed extraordinarily good results with their methods of persuasion, and Venables did follow his patients. But it appears that both he and Ross were more interested in mortality rates and clinical relapses than in the general psychiatric status of their patients at the time of follow-up. Until more evidence to the contrary is presented we at any rate will remain with the impression that in anorexia nervosa psychotherapy offers no advantages over other methods of treatment, even if there is a common psychopathology.

The *immediate results of treatment*, whatever method was used, were not impressive. Only a little over half the patients improved at all, and only one-quarter gained a stone or more in weight during an average stay in hospital of three months. These proportions are in keeping with recent reports, which emphasize the difficulties of treatment. 3 patients died in hospital.

Follow-up Results

Before the present series, the largest case-material studied psychiatrically and followed over a period of years consisted of the 16 patients treated in Dr. S. Cobb's unit at the Massachusetts General Hospital. 14 of them have been described in detail by Nemiah (1950). In this valuable paper, the life histories of the patients can be traced over periods of from three to eighteen years. 4 of the original 16 died, and 6 did not do well. In another study, McCullagh and Tupper (1940) were able to follow the lives of 13 of their 27 patients for from five to ten years. One died and 5 remained ill. In contrast to these rather pessimistic reports, Venables (1930) followed 8 of his 9 patients for from eighteen months to nine years, and found that none had died or relapsed, and only one was "neurasthenic." In our own material, we have been particularly interested in the *mortality rate*, the *incidence of psychosis*, and the *clinical state* presenting after the relief of the anorexia.

Length of follow-up.—25 patients have been traced for five years or more after the onset of their illness, and 5 are more recent cases. In 16, the illness began ten years ago or longer. A tendency to develop psychosis should now be apparent; and we are able to get a good over-all view of subsequent events. Only 5 patients remain untraced.

Fig. 3 shows the results, in broad terms. Out of the original 38 patients, 6 have now died as a result of the illness, 3 while still in hospital and 3 others within three years. The causes of death were: tuberculosis in 2, and circulatory collapse, bronchopneumonia, suicide, and coronary thrombosis each in one patient; the last occurred in a woman aged 25 during appendicectomy carried out when severe emaciation was still present. 2 others have died from diseases unrelated to anorexia nervosa.

The remaining patients have been divided into three groups on the basis of their over-all adjustment. 7 are regarded as still definitely ill; they are underweight, anorexic or vomit easily, and cannot lead normal lives. 2 have had obsessive-compulsive symptoms intermittently; and 2 suffer from severe depressive symptoms.

4 patients are considered to have recovered. 3 are married and have children, and the fourth is working regularly and has no symptoms. "Adjustment", of course, is a relative term: these 4 patients still have some difficulties, but would probably be assessed as normal, in any practical classification.

The main group consists of patients who have made a partial adjustment. All have neurotic symptoms, such as headaches, depressive spells, fear and anxieties without adequate cause, or difficulties in interpersonal relations. They are able to lead fairly normal, though often restricted, lives. Their weights tend to be low, or to fluctuate; their appetites are sometimes pernickety.

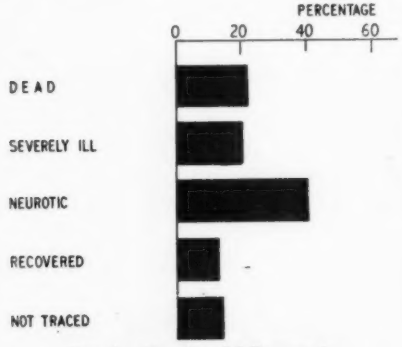

Fig. 3.—Results of follow-up in 38 cases of anorexia nervosa.

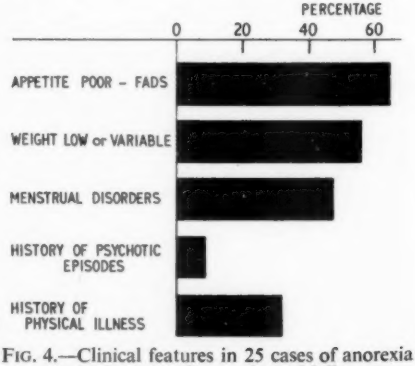

Fig. 4.—Clinical features in 25 cases of anorexia nervosa who were alive at time of follow-up.

Fig. 4 shows the present state of the patients' *eating habits, weights* and *menstrual functions.* It can be seen that the definitive symptoms tend to persist. 65% have some disturbance of appetite, which often takes the form of peculiarities. For example, one patient can suck sweets only in the dark, or after an alcoholic stimulant; another has become vegetarian, because meat is distasteful.

The average weight of the group is now 8 st. There are still many underweight; in fact, in over half, the weight is either low, or fluctuates over a range of 1½ to 2 st.

Menstruation remains disturbed in 45% (11 out of 24 women). 2 other patients have passed through a long period of amenorrhœa, but now menstruate normally. Because of the interest attaching to functional menstruation disturbances, about which little has been written in psychiatric literature, it is worth saying a little more about the subject here.

Of the 5 patients with *persisting amenorrhœa,* 4 remain ill and underweight. Among these, undernutrition seems to be an adequate explanation. Only one patient has done well, yet remains amenorrhœic, seventeen years after the onset of the illness. She alone lends any support to the idea that permanent damage may be done to the endocrine system during anorexia nervosa. It is of interest that this patient was amenorrhœic for two years before the beginning of the illness proper, so that the persisting amenorrhœa is not likely to be due solely to the period of starvation.

In contrast to this patient, is one who has been undernourished, although never cachectic, for many years. During her whole illness, there has been only one brief spell of amenorrhœa. We conclude then that there are other factors beside inanition determining the persistence or otherwise of amenorrhœa.

4 patients have had *irregular menstruation* over years. As far as we can judge, without detailed menstrual diaries, there is, in these patients, a reciprocal relationship between the menstrual and the emotional states. So that, even in the absence of definite clinical relapse, there may be periods of amenorrhœa associated with psychological disturbance. This relationship needs to be studied further, both in patients with anorexia, and in others.

Irregular menstruation may also be a stage on the way to recovery. A woman of 21 was amenorrhœic for eight years, then had irregular bleeding. She was thin and irritable. After marrying, she gained weight, and decided she wanted a child. Regular menstruation followed a minor gynæcological operation and endocrine treatment. She conceived, and now has a son. Thus normal menstrual and reproductive functions may return even after many years' amenorrhœa. This case also raises the question of an endocrine cause of amenorrhœa. But, as a rule, patients who regained normal weight recovered their menstrual function spontaneously.

Psychosis

This was uncommon. No patient has become a chronic inmate of a mental hospital. Psychotic episodes, when they occurred, were short-lived or atypical. 3 patients were at some stage of their illness described as "hypomanic", apparently on the grounds of their hyperactivity. The occurrence of depressive symptoms has already been remarked. No patient has developed a depressive illness who did not show depressive symptomatology from the outset.

Only one patient has been diagnosed as schizophrenic, and, in her, depressive symptoms were always prominent. She was admitted three times to mental hospitals, on one of these occasions experiencing auditory hallucinations and passivity feelings. She responded rapidly to E.C.T. She has since married, and now, nine years after the onset of her illness, weighs 7¾ st. and menstruates regularly. From time to time this patient placed herself on a diet to avoid becoming fat. One of the cases reported by Nemiah later became schizophrenic, and another had an atypical catatonic episode.

Physical Illness

In concluding this review of the morbidity of the group, the incidence of tuberculosis is worth pointing out. 2 patients it will be remembered died from this disease, and 5 others have been subsequently treated for it. It does seem probable that among the tuberculous population there are a number of patients who do not eat for psychological reasons. This may not be recognized, and their low weights wrongly attributed to the physical illness.

Summary

We may summarize our findings as follows: (1) Patients referred to psychiatric clinics, who show symptoms consisting of marked aversion for food, loss of weight, and in women, amenorrhœa, tend to have a poor prognosis; (2) when clear-cut psychotic features are not present from the outset, there is little tendency to develop a psychosis; (3) only 10–20% recover to the stage of good adjustment, the majority continuing to show neurotic symptoms. Many patients still show weight fluctuations and menstrual disturbances, even after five years or more. About 15% die. (4) Our patients, apart from the definitive symptoms, did not exhibit uniformity in their general psychiatric symptomatology. We are not convinced that we have been dealing

with a specific entity. The term anorexia nervosa draws attention to but one aspect of certain psychological disorders which centre around the intake of food. Interest has been focused on this aspect, and on the amenorrhœa, to the neglect of the other symptoms which are always to be found; (5) we have not been impressed by the usefulness of the concept of anorexia nervosa as a psychosomatic entity. As far as we know, the course of the illness is not influenced by endocrine therapy; (6) psychotherapy has given poor results, as have other methods of treatment; (7) we believe that this survey points to the need for further studies into the condition called anorexia nervosa, particularly, into the mechanism and psychological relationships of the amenorrhœa, the effects of different kinds of therapy, and the prognosis in other groups of anorexic patients, especially those treated in general hospitals.

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Sir Paul Mallinson reported that 3 women in the Psychiatric Unit of St. George's Hospital had had various forms of pre-frontal leucotomy. The first was a schizophrenic aged 28 at the time of the operation, who had been ill for seven years; in hospital before operation she weighed 5 st. 13 lb. She had a bilateral standard leucotomy four and a quarter years ago and has been working for the past four years as a mobile physiotherapist, is eating normally apart from a dislike of meat and fatty foods, and although still shy and self-depreciatory, can be regarded as having made a good recovery considering the severity of her illness. Her menstrual periods returned after an absence of six years and she now weighs 7 st. 13 lb.

The second patient was 42 years old at operation and had been ill for over twenty years. This was a patient of obsessional personality but free from overt obsessional symptoms apart from her chronic anorexia; on admission to the Unit, she weighed 4 st. 13 lb. She had a bilateral open rostral leucotomy three years ago. For the past two years and eight months she had been working at her old job, her appetite is good, she weighs 9 st. and considers herself quite well.

The third patient was aged 27 at operation and was a severe obsessional with disabling hand-washing rituals, distressing sadistic fantasies and severe anorexia. She had had fifteen months' analysis without benefit and had been ill for four years before operation; on admission she weighed 7 st. 3 lb. For the past two and a half years she has been fully employed and leading a normal life. Her weight has shown remarkable fluctuations between 9 st. and 11 st. 6 lb. since the operation; she has an abnormally large appetite and has some difficulty in keeping down to her present weight of 10½ st. Menstruation after an absence of four years is regular and normal. She is regarded by her parents and by herself as a perfectly healthy, happy girl.

The speaker pointed out that all 3 patients were completely incapacitated by severe illnesses of long duration for which many different forms of treatment had previously been tried. All 3 patients were of superior intelligence. They were not selected cases, in that no other patients with anorexia nervosa had been subjected to leucotomy in the Unit. Since the results in all 3 cases could be considered to be remarkably satisfactory, he had no doubt that the various forms of pre-frontal leucotomy had a place in the treatment of this disorder.

Dr. D. E. Sands, discussing the treatment of anorexia nervosa by leucotomy, described 2 female patients of 26 and 43 years, known personally to him. A further female patient of 36 years was quoted from the literature (Carmody, J. T. B., and Vibber, F. L. (1952) *Ann. intern. Med.*, **36**, 647). In these 3 cases duration of illness ranged from nine to twenty years, and before operation all 3 showed dependent, retiring types of personality and were emotionally immature. The first 2 patients both had parents whose influence in early life was ill-balanced, one case having a very dominating father and the other a dominating mother. The first of these 2 married an over-anxious and over-protective husband. Both patients were over-anxious for security in adult life, and used the illness situation for personal gain.

In all 3 cases there was a decisive improvement, both mentally and physically, after rostral or lower orbital bilateral operations. All were able to return to home and eventually to work. One has since shown partial relapse of neurotic but not anorexic symptoms. In one case a post-leucotomy syndrome with temporary antisocial conduct delayed her return to full occupation.

It is suggested that in these chronic types of anorexia nervosa, where no progress has been made by other methods of treatment over the course of some years, leucotomy might be considered earlier than was the case in these 3 patients, to the advantage of both patient and relatives. The rostral or lower orbital incisions appear to be the operations of choice in the first instance.

Section of Pædiatrics

President—D. W. WINNICOTT, M.A., F.R.C.P., M.R.C.S.

[*February* 27, 1953]

Symptom Tolerance in Pædiatrics

PRESIDENT'S ADDRESS

By D. W. WINNICOTT, M.A., F.R.C.P., M.R.C.S.

MY theme, which is foreshadowed in the title, might lead along two distinct paths. I mention one of these only because I may have been expected to follow it. I refer to the fact that the natural bodily processes tending towards health and towards a resolution of illness have become more than a little obscured by the recent flood of advances in chemotherapy. It is indeed difficult for a House Physician at the present time to find out by experience what a child does with pneumonia with no more help than the good nursing which, thirty years ago, was the only treatment. To-day, and we all agree about this, even a boil must not be allowed to take care of itself. I feel that in the best of the medical schools the teaching does include a reminder that children lived through illnesses before penicillin and that even to-day it is the child and the living tissues that ultimately bring about a restoration to health, not the antibiotic.

I have not followed this important trail because this theme has been developed with great competence in various addresses to medical students by physicians who remember the bad old times, and who see that from the teaching point of view the bad old times could claim some points in their favour. My theme follows another path which in the end will, I think, prove to be a related one, since again it concerns the natural tendency towards health and the use that we can make of this tendency as doctors. In the psychological field the principle that there is a natural tendency towards health or developmental maturity is one which has a particular significance. It could be said that much of physical disease is due to an invasion from the environment or to an environmental deficiency, and is not a purely developmental disorder. By contrast, psychological disorder can always be described in terms of emotional development either delayed or distorted or in some other way prevented from reaching the maturity that is due at the age which the child has reached. There is an even closer link therefore in psychological medicine between the normal and the abnormal than there is between physiology and the pathological processes of tissues and functions. In fact when there is only a disturbance of physiology then the illness is usually psychogenic.

When I was thinking over the relationship between pædiatrics and child psychiatry it occurred to me that this relationship involves not only a difference between the fields but also a difference of emotional attitude between those who adopt the one or the other approach to a case. The pædiatrician feels the symptom as a challenge to his therapeutic armoury. It is hoped that this will always be true. If a child has a pain, then the sooner it is diagnosed and the cause removed the better. By contrast, the child-psychiatrist sees in the symptom an organization of extreme complexity, one that is produced and maintained because of its value. The child needs the symptom because of some hitch in emotional development.

(For the purposes of clear argument it is helpful to assume that our physically ill child is psychiatrically healthy, and that our psychiatrically ill child has a well body. Although this is very often not true it is a justifiable simplification for us to make just now.)

THE CHILD NEEDS THE SYMPTOM

The psychiatrist is therefore not a symptom-curer; he recognizes the symptom as an S O S call that justifies a full investigation of the history of the child's emotional development, personal, and relative to the personal environment and to the culture. Treatment is directed towards relieving the child of the need to send out the S O S.

There is, as I have already indicated, a degree of artificiality in this statement of contrasts. The best body-doctors do also seek causes and, when possible, employ as their chief therapeutic the natural tendency of the body to be healthy. But even body-doctors who are suitably tolerant of symptoms that are physically determined and who seek first causes when faced with physical disease, tend to become symptom-allergic before a syndrome of psychological ætiology. They develop an urge to cure the moment they are confronted with an hysterical conversion symptom, or a phobia that has no apparent sense in it, or a sensitivity to noise that seems quite mad, or an obsessional ritual, a regression in behaviour, a mood disorder, an antisocial tendency, or a restlessness that connotes a hopeless confusional state at the core of the child's personality.

I am convinced that symptom intolerance appears simply because the body-pædiatrician does not know anything much about the science called dynamic psychology (psycho-analysis for me), and yet by this science alone can sense be made out of symptoms. Seeing that this fifty-year-old science is at least as large as physiology, and includes the whole study of the developing human personality in its setting, it is not to be wondered at if the weary postgraduate, having reached the heights of a pædiatric registrarship, boggles at yet another discipline, and eschews the new training that alone qualifies for psycho-therapeutic practice.

This problem of the double training must be left to solve itself in the course of time; meanwhile we must expect and welcome the two types of approach, the physical and the psychological, and we must try to assimilate the contribution that each can make to pædiatrics.

Unfortunately I must now narrow down my subject, which could be almost infinite in width and breadth. I have chosen to discuss enuresis, although I confess I find it difficult to leave aside so very much that would interest both myself and any pædiatric audience.

Enuresis: An Example

There are enuresis clinics run by pædiatricians, and usually the avowed aim in such clinics is the cure of the symptom. Mothers and children are grateful. There is nothing to be said against such clinics except that they sidetrack the whole issue of ætiology, of enuresis as a symptom that means something, as a persisting infantile relationship that has value in the economy of the child. In most cases cure of the symptom does no harm, and when a cure *could* do harm the child usually manages, through unconscious processes, either to resist cure or to adopt an alternative S O S sign, one that produces transfer to another type of clinic.

While these pædiatric clinics are coming and going, child psychiatrists are all the time meeting with the symptom enuresis, and often the symptom is easily seen to be quite a subsidiary phenomenon, a little bit of the huge problem of a human being engaged in trying to develop to maturity in spite of handicaps.

I shall give only one case out of the many hundreds to hand; by this one case description I shall hope to show how enuresis appeared in the course of a psychiatric illness.

I choose that of a boy for whom psycho-analysis was not available, yet whose cure (if I may call it a cure) depended, in part, on two psycho-therapeutic sessions.

At these two sessions the boy was drawing all the time, and I was able to take notes except at the most critical moments when feeling was so tense that the taking of notes would have done harm.

The case, not an unusual one, is the more suitable for presentation, because the child's treatment was mainly carried out by the parents, who were able to mend their home that had been broken by the war; I hope that you will be able to see in this part of the description, where the parents did the work, a type of illness and recovery that you have watched yourselves in those of your own child patients who have been able to use a period of physical illness as an opportunity for making delayed growth in personality. This boy was lucky; he was able to get what he needed without the otherwise helpful physical illness.

The Case. "Philip"

Philip, aged 9, was one of 3 children in a good family. The father had been away over a long period during the war, and at the end of the war he retired from the Army and settled down to rebuild his home, starting to be a farmer in a small way. The two boys were at a well-known prep school. The headmaster of the school wrote (October 1947) to the parents to say that he must advise them to take Philip away because, although he had never thought of the boy as in any way abnormal, he had found him to be the cause of an epidemic of stealing. He said "I can easily deal with the epidemic if Philip is removed"; and he wisely saw that Philip was ill and would not be able to respond in a simple way to corrective treatment. As a result of this letter, which came as a great shock to the parents, they consulted their general practitioner and he, on the recommendation of a psychiatrist, referred the case to me.

Consideration of these details shows how precarious is this matter of the referral of a psychiatrically ill child before damage is done. It was almost by chance that I was in a good position to give help at the very beginning and before a moral attitude towards the boy's delinquency had had time to become organized, and before symptom intolerance had developed to the extent of producing panic therapy.

I arranged first of all to see the mother and in a long interview I was able to take the following detailed history. This history turned out to be substantially correct, although in one important detail the exact truth did not emerge until I had an interview with the boy.

Parents.—I was able to gather that the father and mother had the ability to form and to maintain a good home, but that the disturbances of the war had caused serious disruption which affected Philip more than the brother. The small sister was obviously developing normally and was able to derive full benefit from the mended home. The parents were inclined towards spiritualism but they made it clear to me that they were not trying to impose their way of thinking on the children. The mother had a dislike of psychology and claimed to know nothing about it, and this proved to be valuable to me in my management of the case, since I was able to rely on her feelings and on her native or intuitive understanding of human nature and not on her sporadic reading and thinking.

Early history.—The brother was breast fed for five months and was a straightforward personality from the beginning. He was much admired by Philip.

Philip's birth was very difficult; the mother remembers it as a long struggle. The amniotic fluid broke through ten days before the birth, and from the mother's point of view the birth started and stopped twice before the boy was actually born, under chloroform. Philip was fed at the breast for six weeks; there was no initial loss of weight, and there was an easy transfer to the bottle. As a baby he was what is usually called bonny, until the age of 2, when the war started to interfere with his life. From this age he had no nursery days at home and he developed into rather a quiet child, perhaps too easy to manage. Nursery life had to be shared with tough and strange children. At this point he became excessively catarrhal and he developed an inability to try to blow his nose. The catarrhal tendency has continued and was not favourably affected by tonsillectomy at 6 years. The mother is subject to asthma and she thinks that the boy occasionally had asthma of slight degree. The mother looked after Philip most of the time although with the help of a nanny, and she early noticed the difference between the two boys. Not only was Philip less healthy on account of the catarrh, but also his co-ordination was poor.

Effect of the war years.—When Philip was 2–4 years old he and his brother were away with the mother, after which they were back in the original house. The home, however, which was broken up when he was 2, was not restored until the father retired from the Army, not long before the date of the consultation. The children's possessions were necessarily scattered, never all available at one time, and any one object was liable to become lost. As compared with the brother, Philip was not demonstrative. He was, however, affectionate enough with his mother and sister. The mother felt him to be a stranger and what possessions he had were very private to him. Actual difficulties did not show, however, till he was 6 years old.

In regard to what is called toilet training, he had been easy, and bed-wetting had never been a trouble.

Original thieving.—When Philip was 6, which the mother reminded me was the time of his tonsillectomy, he came back home with the nurse's watch. In the course of the next three years he stole another watch and he also stole money which he would always spend. Other objects were stolen and they always got damaged. He was not without money of his own and he developed a passion for book collecting. Being very intelligent and a good reader, he actually read the books that he bought, but the buying of the books was very important to him. They were mostly small books on moths and grasses and dogs, books of classificatory type. It was noticed that he paid 15s. for a small book on ships without seeming at all to recognize that this was expensive. Along with these symptoms the parents had noticed a change in the boy's character but they could not easily describe it. The parents were really disturbed when the following incident occurred: staying at a house, on the way home from a holiday, he stole a car registration book that belonged to the people that owned the house. He did not attempt to hide it and the parents put down the theft to his undoubted love of documents of all kinds. On looking back they could see that at this time he began to become an untidy person. Moreover he became less and less interested in possessions except new books, and along with this was an exaggeration of the wish to give things away to his sister of whom he was very fond.

This was in the period when he was 6–8, up to the year in which he was brought into my care.

Birth of sister.—At the birth of the sister (when he was 6) the mother said that he was first of all disturbed and openly jealous but he soon became fond of her and returned to a fairly good relation with the mother, not as easy, however, as before the birth. At this time the father began to discover that his children were interesting for the first time, partly because he had a daughter and mostly because he was now able to be living in his own home more and more. Incidentally, the mother told me that both she and the father had longed for a girl for their second child. When Philip appeared they took some time to adjust to the idea of another boy. The birth of the daughter eventually brought great relief to the family and undoubtedly released Philip from a vague feeling that he was wanted in some way to be unlike what he was. I made a special note that the tonsillectomy, which had apparently brought about the change in this boy's personality, had been done soon after the birth of the sister, and later I was to discover that the birth of the sister was the more important disturbance. At this time, the boy being 8, he became shy of anything that might make people laugh at him. The mother reported as an example that he had a swelling on the face caused by a sting. Rather than risk being laughed at he would be excessively tired and would stay in bed. In defence

against being laughed at he developed the art of mimicry and in this way he became able to make people laugh at will. He also developed a fund of amusing stories, and in this way he further warded off derision. While telling me this the mother felt in despair as she recognized while talking how much she had been at sea in her dealings with this boy while she had not had difficulty in understanding the brother and the sister. She had the ability to be closely in touch with a normal child but not the ability to keep in touch with a child who was ill, and it was important for me to recognize this as I needed her co-operation. Later on I described what the boy needed of her not in terms of the needs of a psychiatric case but in terms of the need of a normal infant, explaining that the boy would need to be allowed to go back and to be an infant in relation to her, thus making use of his newly founded home. Thus I avoided having to instruct her against her will in psychopathology.

Sleep.—Sleep was always disturbed by nasal obstruction. Philip would wake and require his mother to help and it is likely that he was able to use this physical difficulty, without knowing it, of course, in order to get his mother's presence at night; had he not had the nasal obstruction the mother would have been brought to his bedside by nightmares or some phobia. He had a phobia of being hurt and after tonsillectomy he had a phobia of doctors.

When I asked what happened when he got excited, did he get ill or just jump around?—the mother said, "When you expect him to get excited he becomes quiet and retiring and asks repeatedly 'Oh what shall I do? What is there to do?' " The mother had noticed that it was important to him that he should be alone for some part of every day. He could make use of distractions, and for instance when taken to Switzerland he soon learned to ski, but more by effort of will than by natural skill.

The mother reported that he had bouts of increased urgency and frequency of micturition which she connected with the nasal obstruction. At the school the boy was considered to be healthy and the nasal obstruction seemed to be less in evidence. I went with the mother and boy to an oto-rhinologist who gave valuable specialist advice, but also ordered a whole host of symptom relievers from which the boy had to be rescued.

At school Philip was thought to be intelligent but lazy. The headmaster gave him a bad report but in a letter to me he said that he had never thought of this boy as being in any way abnormal until the stealing started. He was not unaccustomed to laziness and expected the boy to make good in the end. From this detail I think it can be seen how a really good school can miss psychiatric illness.

Philip was fond of the country. He possessed a greyhound of his own and this turned out to be very important, playing a vital part in his cure. While he was in trouble at school he wrote a letter in which there was no indication that he was in distress.

Summary of case history.—This history which the mother was able to give me showed that the boy started life well but that there was a disturbance in the child's emotional development dating from 2 years. In defence against environmental uncertainty he became withdrawn and relatively unco-ordinated. At 6 he started a degeneration of the personality which was progressive, leading up to the major symptomatology at 9 for which he was brought to me.

Management.—Although I had not seen the boy I was able to begin on the arrangement of his management. It was clear that psycho-analysis was out of the question, since a daily journey to London or even a weekly one would be a disturbance of the use that the boy could make of his mended home, and it would be this mended home that would carry the major burden of the therapy.

I told the mother that this boy would need her help as it was clear that he had missed something at the age of 2 years and he would have to go back and look for it. She quickly understood, and said, "Oh well, if he has to become an infant, let him come home, and as long as you help me to understand what is happening I can manage". She proved that this was no idle boast and eventually she was able to take the credit for having brought the child through a mental illness; the home provided the mental hospital that this boy needed, an asylum in the true sense of the word.

In technical terms, the boy regressed. He went back in his emotional development in a way that I shall describe presently, and then came forward again. It was at the depth of this regression that the bed-wetting appeared which is the link between this case and the main theme of my Address.

Interview with Philip

My next task was to see the boy. I needed this interview in order to know where I stood in the management of the case over the next few months (mostly by telephone), and also because the boy was ready for insight which he got in this one and a half hours and which, although not psycho-analysis, was certainly the application of knowledge gained in psycho-analytic work.

First interview with Philip.—There was no initial difficulty. The boy was an attractive intelligent lad, rather withdrawn, not showing much evidence of making objective observations about me. He was evidently preoccupied with his own affairs and slightly bemused. His sister came with him and he behaved in a natural way with her, and easily left her with her mother while he and I went into the playroom. I adopted a technique which suits these cases, a kind of projection test in which I play my part. Figs. 1–8 show the results of this. It is a game in which first I make a squiggle and he turns it into something, and then he makes a squiggle and I turn it into something.

(1) *My* squiggle (Fig. 1). He turned it round and quickly called it a map of England, adding a line which was needed in the region of Cornwall.

I saw immediately from this that he was in a highly imaginative state, and that I would get results that were very personal since this squiggle could have been made into almost anything.

(2) *His* squiggle, and I delayed making it into anything myself, thus giving him a chance to display again his imaginative capacity. He immediately said it was a rope going up into the air and he indicated the air by thin strokes crossing thick strokes of the rope.

(3) *He* again made a squiggle (Fig. 2) and I quickly turned it into a face which he called a fish. Again this was an indication that he was preoccupied with his personal or inner reality and not particularly concerned with being objective.

(4) *My* squiggle (Fig. 3). It was astonishing to witness the way in which he immediately saw in it a mother and baby sea-lion. As events turned out it was justifiable to understand from this drawing that the boy had a powerful maternal identification; also that the mother-baby relationship was of especial importance to him. Moreover this picture has beauty, not indeed on account of the squiggle, but on account of his use of it.

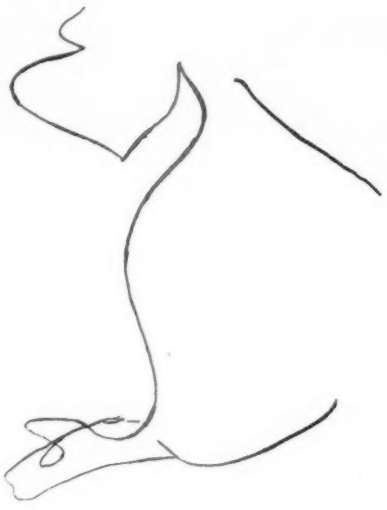

FIG. 1.

FIG. 2.

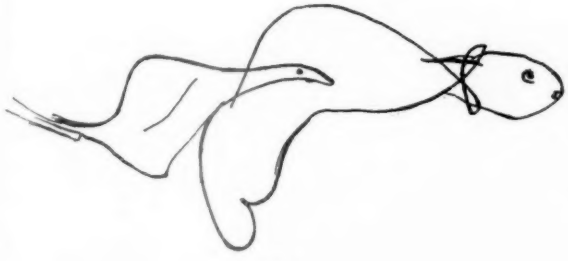

FIG. 3.

FIG. 1.—My squiggle. His modification. His comment—England.

FIG. 2.—His squiggle. My modification. His comment—a fish.

FIG. 3.—My squiggle. His modification. His comment—a sea-lion with a baby.

(5) *His* squiggle. Before I could make anything of it he had turned it into men roped together climbing rocks. This belonged to his recent experiences in Switzerland.

(6) *His* squiggle again, which he interpreted as a small whirlpool with waves and water. For him this was quite clear, though not so clear to me.

(7) *His* squiggle again, which he turned into a boot in water, again something absolutely personal to himself. I had already recognized that he was almost in what could be called a sleep-walking state, and this prepared me for the psychotic features about which I was to learn a little later on.

(8) *My* squiggle (Fig. 4). He immediately turned it into what he called Punch with tears in his clothes.

He was now very much alive in a creative sense and he said "There are tears in his clothes because he has been doing something with a crocodile, something dreadful, probably annoying it, and if you annoy crocodiles you are in danger of being eaten".

(9) He was now talking about dream material and I was therefore in a position to investigate his dreams. I spoke about the frightening things that could occur to him in his thoughts, whereupon he drew Fig. 5 which he called a wizard. There was a long story with this. The wizard turned up at midnight at school. Apparently he did a lot of watching for the wizard in the night. This wizard had absolute power and magical power. He could put you underground and turn you into things. This wizard turned out to be an important clue in the understanding of the compulsion to steal.

He was now willing to tell me dreams. He was going in a car with his mother. The car was going downhill. There was a ditch at the bottom of the hill and the car was going so fast that it could not possibly stop. At the critical moment magic happened, good magic, and the car went over the ditch without falling into it.

I put into words what was implied by this and by the way that he told it to me. I said that he was frightened, that in the dream he had had to use good magic because this meant that he had to believe in magic and if there was good magic there was also bad magic. His inability to deal with reality and the necessity to employ magic was the awful thing.

FIG. 4.—My squiggle. His modification. His comment—Mr. Punch with tears in his clothes.

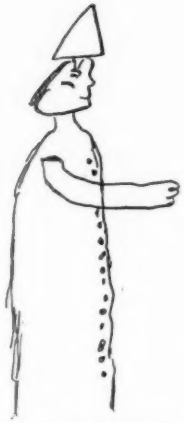

FIG. 5.—Drawing No. 9. The wizard.

He told me a further dream. He had hit his headmaster in the tummy, "But the headmaster is nice", he said: "he is a man you can talk to". I asked him if he was ever sad and he was now communicating with me from very deep in his nature when he said that he certainly knew what sadness meant. He had a name for it. He called it "dreary times". He said that the worst sadness happened a long time ago and he then told me about his first separation from his mother. I was not sure at first what was his age at the time of the experience. He told me: "Mother went away. I and my brother had to live by ourselves. We went to stay with my aunt and uncle. The awful thing that happened there was that I would see my mother cooking in her blue dress and I would run up to her but when I got there she would suddenly change and it would be my aunt in a different coloured dress."

He was telling me that he was constantly hallucinating his mother, employing magic but constantly suffering from the shock of disillusionment. I spoke to him about the awfulness of finding things you thought real were not real. He then drew a mirage (10) and took a holiday from the subject of hallucinations by giving the scientific explanation of a mirage. His uncle had told him all about it. "You see lovely blue trees when there are really no trees there."

He also said that he was very fond of beautiful things. "Now my brother, he thinks of nothing but ships and of sailing, and that is quite different. I love beauty and animals and I like drawing." I pointed out to him that the beauty of the mirage had a link with his feelings about his mother, a fact which I deduced from the blue colour of the mirage and of the mother's frock.

My notes at this point become less clear as the situation had become very tense and the boy had become deeply serious and thoughtful. He told me quite spontaneously about his depression, or what he called dreary times. It now turned out that the worst dreary time had occurred when he was nearly 6 and I was now able to see the importance of the birth of the sister. By his mother's going away he had meant that she had gone to a nursing home to have the child. It was then that he and his brother stayed with the aunt and uncle and although the brother was able to manage easily, Philip only just managed to keep the thread of experience unbroken.

He was not only hallucinating but also he was needing to be told exactly what to do and his uncle, recognizing this, had deliberately adopted a sergeant-major attitude and by dominating the boy's life he had counteracted the emptiness which resulted from the loss of the mother. There was one other thing that kept him going, which was that his brother, who was a great help, constantly said: "It will end; it will end."

The boy now had the first opportunity he had ever had for talking about his real difficulty at the time, which was to come to terms with the mother's capacity to have a baby, which made him acutely jealous of her. The picture of the mother and baby sea-lion showed how much he had idealized the mother-infant relationship. The fact that the baby had turned out to be a girl had been a relief to him.

He said, "I spent all the time thinking of how soon the end would come, or else I just felt sick". Once at school he had felt homesick, which was another kind of dreariness or depression, and he went to the headmaster. He said: "The headmaster tried everything but he could not help." He then compared the headmaster with me and quite openly said that, whereas the headmaster had only been able to say "Cheer up" I had been able to give him some understanding, of which he was in great need.

We were now able to return to the wizard who turned out to have the overcoat of his soldier uncle who had dominated his life, thus saving him from the emptiness of depression. He now told me that the wizard had a voice which was exactly that of the uncle. At school this voice continued to dominate him. The voice told him to steal and he was compelled to steal. If he hesitated the voice would say: "Don't be a coward; remember your name." "In our family there are no cowards." He then told me about the main episode for which he had been expelled. A boy had said to him, "Oh well, there's nothing very much about what you've done; anybody might have stolen pound notes and things like that. It isn't as if you had stolen poison drugs from the Matron's cupboard." After this the voice of the wizard told him that he must take poison drugs from the Matron's cupboard and he did this with consummate skill. It was when he was found to be in possession of dangerous drugs that he was expelled, but for him there was no shame since he had obeyed the voice and had not been a coward. Moreover I would add that in stealing he was in the direction of finding the mother that he had lost, but this is another subject which cannot be followed up here.

I have tried to give a faithful account of the interview but I cannot hope to convey the feeling of something having happened which was very real to both of us.

I was somewhat exhausted and ready to stop but he sat down to one final picture (Fig. 6).

FIG. 6.—Drawing No. 11. "His father in a boat; over the boat is an eagle and the eagle is carrying a baby rabbit".

(11) After drawing in silence he said that this was his father in a boat. Over the boat is an eagle and the eagle is carrying a baby rabbit.

In the setting it was clear that Philip was drawing not only to "seal off" the interview but also to report progress. I started to put this into words. I said that the eagle stealing the baby rabbit represented his own wish or dream at the time of maximum distress, to steal the baby sister from the mother. He was first of all jealous of the mother for being able to have a baby by the father, and also he was jealous of the baby since he had an acute need to be a baby and to have a second chance to make use of his mother in a dependent state. (Of course, I used language appropriate to his capacity to understand.) He took up the theme, and he said: "And there's father, all unconcerned." You will remember that his father had been overseas. The fact that his father was away fighting for his country is now of great importance to him, and he can boast of this at school. But in respect of his childhood needs the father was neglecting his son's urgent need for a father actually on the spot, friendly, strong, understanding, and taking responsibility. But for his uncle and his brother he would have been sunk when his relation to his mother was interrupted by the separation and by his jealousy of her. The boy was now ready to go.

Second Interview.—I saw him again within a week and I shall not need to give a detailed account. I show his drawing, however (Fig. 7), through which he announced that the wizard and his voice had gone since the first interview. In this drawing of the wizard's house I am in the wizard's house with a gun, and the wizard is retreating. The smoke indicates that the wizard's wife is in the kitchen cooking. I go in and take the magic from her. You will remember his need to find his mother cooking—the alternative was the witch, and the cauldron, and the magic spells of the woman of early infancy, who is terrible to think of in retrospect, because, of the infant's absolute dependence. This already has some flavour of fantasying, of operation at a less deep level and indeed the tenseness of the atmosphere of the first interview was missing. I was no longer within the inner circle of the child's personal and magical world, but a person listening to him talking and hearing about his fantasies.

Two more drawings:

One shows the wizard going down the school corridors. After this he told me again about the hallucination of the mother who changed to the aunt when he put what he saw to the test. Also here there was an important bit about the way in which the wizard's candle belonged to genital erection and to ideas of fellatio and ideas of burning hair. I had to be able to take this, along with the magic and whatever else might turn up, else I would suddenly intrude myself, as much by any display of prudery or blindness as by an active introduction of my own ideas. But this was not to be an analysis and I had to avoid giving understanding in relation to the repressed unconscious. One learns to keep a sensitive balance in doing this work—comparable with the skills that are acquired by those whose practice takes them to cisternal punctures or to the transfusing of the newly born.

The last drawing of the second series (Fig. 8) shows the wizard again. This time the wizard is being laughed at. It is a "funny" picture. It will be remembered that it was part of the child's illness that he expected to be laughed at; the object of the derision had now been got outside the house (himself), and in the place of the wizard was his highly subjective idea of me. I was simply a person who fits in and understands, and who verbalizes the material of the play. In verbalizing I talk to a conscious self and acknowledge the PLACE WHERE FROM in his total personality, the central spot of his ENTITY, without which there is no HE.

FIG. 7.—The wizard's house.

FIG. 8.—A "funny" picture of the wizard.

Figs. 1 to 8 have been selected out of 14 drawings.

The Third Interview.—This started with a drawing in which his enemy drops a knife on his greyhound. The enemy is the son of the uncle who played such an important role in his life during the depression and whose voice and Army overcoat supplied the details for the wizard that he employed to counteract his "dreariness". This cousin was hated because of Philip's powerful love of the cousin's father.

This third interview changed into an ordinary play hour, and I just sat and watched Philip make a complex plan with my train set rails. Whenever he came to me subsequently he just played with the train, and I did no more psychotherapy. Indeed I must not, unless I had been able to let the treatment develop into a psychoanalytic treatment, with its reliable daily session arranged to last over a period of one, two, or three years. This was never contemplated in Philip's case.

THE ILLNESS AT HOME

I now come to the illness that the boy had to have during which the parents provided him with asylum. It can be briefly described. This child needed my personal help, but at the borderline there are the common cases in which the psycho-therapeutic session can be omitted, and the whole therapy can be carried out by the home. The loss is simply that the child fails to gain insight, and this is by no means always a serious loss.

Philip was accepted at home as a special case, an ill child needing to be allowed to become more ill. By this I mean that there had been a controlled illness and this was to be allowed to come to full

development. He was to receive that which is the right of every infant at the start, a period in which it is natural for the environment to make active adaptation to his needs.

What it looked like was this: Philip gradually became withdrawn and dependent. People said he lived in a fairy world. His mother described how he did not so much get up in the mornings as change from being in bed to out of bed, this simply because someone dressed him. This is a lay method of saying that the boy was in a somnambulistic state. On a few occasions the mother tried encouraging him to get up but he was quickly reduced to tears of distress and she abandoned all encouragements. At meals he gathered the utensils around him and ate alone, although with the family. He seemed uncivilized, taking big gulps of bread, and eating the jam down first. He would eat everything that was there rather mechanically, not seeming to be either able to want or to reach a stage of having had enough. All the meal he was in a preoccupied state.

He went downhill steadily, becoming less and less able to live in his body or interested in his appearance, but he kept in touch with the enjoyment of body by watching his greyhound for hours on end.

His gait became unco-ordinated, and towards the bottom of the regression he progressed by a hop-skip technique, arms waving like windmill sails, or by a series of lurchings, as if propelled by some crude agency living within the self, certainly not walking. While progressing in this way he made noises which his brother called "elephant noises". No remark was ever made about his many oddities and eccentricities and bizarre behaviour patterns. He had the cream from the one cow, and also he figuratively skimmed the cream off the homeliness of the home.

On occasions he would come out of this state for an hour or two, as when the parents gave a cocktail party, and then he quickly returned to it.

Once he went to the local "Hop" and there his queer attitude to girls came to the fore. He danced a little, but only with a very odd and fat creature known as "the galleon", assumed in the locality to be mentally defective. Dick Barton became an obsession during this period, and his life revolved round this and watching the dog.

Then the bottom was reached. He was always tired. He had increasing difficulty in getting up at all. At last I have reached the symptom which made me choose this case for description. He became for the first time since infancy a bed-wetter. The mother got him up, between 3 and 4 a.m. each night, but he was usually wet. He said to her: "I dream so vividly that I have got to the pot." Also at the time he was addicted to water, drinking to excess. Of this he said: "It's such fun, it's delicious, it's good to drink."

All this took about three months.

One morning he wanted to get up. This marked the beginning of his gradual recovery, and there was no looking back. The symptoms peeled off and by the summer (1948) he was ready for a return to school. This was delayed, however, till the autumn term, one year after the start of the acute phase of the illness.

There was no return, after the first psycho-therapeutic interview, either of the wizard or of the voice, or of the stealing.

On the return to the same school Philip picked up quickly, and there was no difficulty about living down a reputation for thieving. Soon the headmaster was able to write the usual letter one expects, asking what was all the fuss about, the boy being perfectly healthy and normal. He seemed to have forgotten that he had expelled him a year previously.

At 12½ Philip went to a well-known public school, rather a tough one, and at 14 he was reported to be 5 ft. 5 in., broad in physique, manly by nature, always out of doors, and good at the usual games. He was reported to be one year ahead of his age group scholastically.

Additional Facts

The treatment cost the parents £15 15s. 0d. which was well within their capacity to pay.

No Intelligence Test or Rorschach Test was done, as this was quite unnecessary.

There was no need for Psychiatric Social Worker help, as the whole problem was well within the personal scope of one person.

Moral

I now come to the moral. If a pædiatrician had been consulted on account of the bed-wetting, what would it have seemed to him had he become involved in the case at the point of the child's maximal regression? Ordinarily the mother would not know what was going on, nor would the child. Possibly in this particular case a sensitive pædiatrician, giving plenty of time to history-taking and to a personal interview, might have been able to place the bed-wetting in its setting along with the compulsive stealing. In the case of Philip there was an exceptionally favourable setting for an illness to develop to its full extent and to come to its natural end. Usually the setting is too complex or insufficiently stable to allow of a clear exercise of the complex processes that could be watched in Philip's case. There is nothing more difficult to treat than the child who is apparently normal except for bed-wetting. In these cases the regression is a brief one recurring at a certain period in the night-time, hidden, but important to the child just as the regression was to this boy that we have studied in greater detail.

(I must put in a note here that I am over-simplifying again since not all cases of enuresis can be described in terms of regression, and beyond the question of regression there is the place of the excretory function as an experience in every infant's life, part of a highly complex development of the capacity for object relationship.)

It would have been futile to have tried to cure Philip's enuresis.

Recapitulation

I do see the point of view of the pædiatrician who, not being concerned specifically with psychology, must ignore the meaning of symptoms and must try to cure them. But I do ask these doctors to give the psychologist credit for his point of view, just as the psychologist gives the pædiatrician credit for special knowledge of infant physiology, the biochemistry of fluid loss, the grouping of blood, and the early diagnosis of brain tumour. The two disciplines should produce different kinds of pædiatrician each with a healthy respect for the other.

I had to keep to one simple thing—enuresis, and indeed to one aspect of this one symptom. We need some organization for the joint study of the developing human being, body and soul; or shall I say, psyche, soma, and intellect?

The phrase "Department of Child Health" (not Pædiatrics) pre-supposes an eventual growth of mutual understanding, one in which the child-psychiatrist can be understood by his pædiatric colleagues even when he shows a maddening degree of symptom tolerance.

BOOK RECEIVED FOR REVIEW

(As no reviewing is undertaken in the "Proceedings" this list is the only acknowledgment made of books received for review)

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